

# INTRODUCTION



LOUIS H. BERMAN, LUCIA BLANCO, AND STEPHEN COHEN

## CHAPTER OUTLINE

### DEFINITION AND CLASSIFICATION OF INJURIES

#### Hard Tissue Injuries

##### Tooth Injuries

Crown Fractures

Root Fractures

Luxation Injuries

##### Alveolar Injuries

#### Soft Tissue Injuries

### ETIOLOGY AND EPIDEMIOLOGY

#### Trends

Cause of Injury

Occurrence of Injury

Gender Significance in Injuries

Type of Injury

Dental Location of Injury

#### Predetermining Factors

### PREVENTION

### CONCLUSION

Dental traumatology is the branch of dentistry that encompasses the epidemiology, etiology, prevention, assessment, diagnosis, and management of trauma to the jaws and surrounding tissues<sup>15,48,50</sup> (Fig. 1-1). It also embraces posttraumatic sequelae, such as root resorption and its treatment<sup>4,13,47</sup> (Figs. 1-2 and 1-3). Because dental trauma can be simple or complex, its management may be interdisciplinary or multidisciplinary. Timely care is as important as the care itself because most adverse posttraumatic sequelae are a consequence of inefficient or inappropriate emergency care.<sup>1,18,21,51</sup>

With this first edition of *A Clinical Guide to Dental Traumatology*, we have presented the comprehensive topics pertaining to dental trauma in an organized and evidence-based approach. Each of the following chapters has been written by leading authorities in the field who have offered their expertise in describing the various types of traumatic dental injuries, with detailed explanations of how to optimally manage the various types of injuries and posttraumatic sequelae. Traumatic injuries are typically *quick, sudden, and unexpected*; this is why clinicians must be prepared to render appropriate emergency care at any time.

Injuries are not necessarily accidents; this is why throughout this text, the term *accident* has been replaced with the terms *incident, injury, or trauma*.<sup>16,30</sup>

Therefore trauma can be divided into two main categories:

- **Nonintentional injury:** includes domestic, recreational, sports, work, vehicular related injuries, and other such injuries that are not inflicted on purpose by one's self or another person.
- **Intentional injury:** includes suicide, homicides, domestic abuse, war, terrorism, and other such injuries that are purposefully inflicted.<sup>30</sup>

VISIT...

LANZAROTE  
*Caliente*.COM



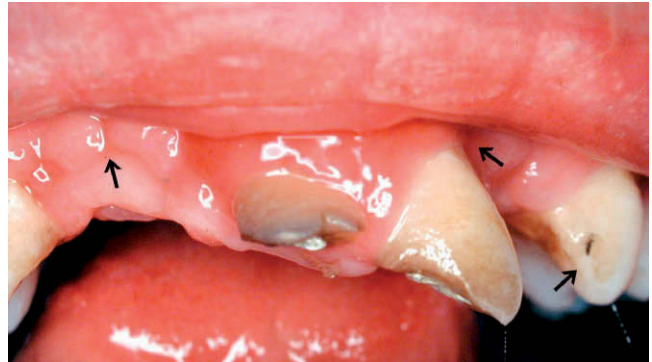
A



B



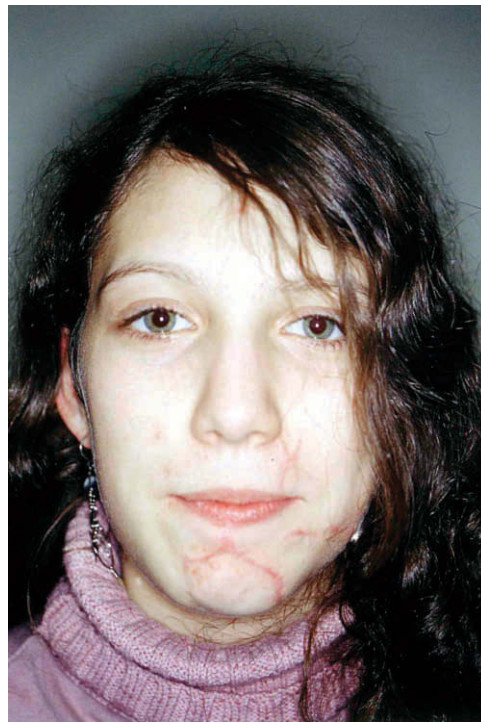
C



D



E



F

**Figure 1-1** Sixteen-year-old female sustained multiple hard and soft tissue injuries following an automobile crash. **A**, Note the extensive scarring 4 months after injury. **B**, Closer view of the mandibular area. **C**, Avulsed maxillary left canine with attached bone found in the automobile. Particles of glass were also found in the bone. **D**, Avulsion of maxillary right incisors. Extrusive luxation of maxillary left central incisor with horizontal fracture of crown cervically. Complicated crown fracture and subluxation of maxillary left lateral incisor. Avulsion of maxillary left canine (arrow). Yellow stains on teeth are secondary to prolonged rinsing with chlorhexidine. Uncomplicated oblique crown fracture in maxillary left first premolar (arrow). Note red scar in mucosa secondary to deep wounds and extensive loss of bone secondary to trauma. **E**, One month posttrauma, panoramic radiograph reveals mandibular fractures with synthetic bone augmenting the alveolar defects resulting from avulsed teeth. Note that the patient also suffered avulsion of the lower right lateral incisor and canine. **F**, One year after the initial consult. Plastic surgery is scheduled to minimize the scarring and disfigurement.





A



B

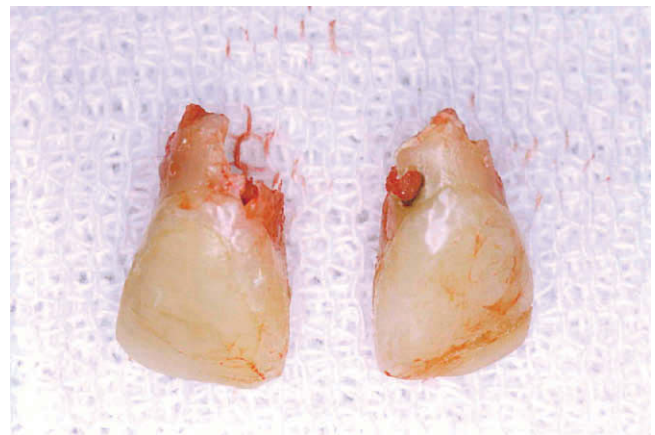
**Figure 1-2** Fourteen-year-old male fell, hit his mouth, but did not report to dental office until 6 months after trauma. **A**, Lateral luxation of maxillary right lateral incisor, extrusive luxation of maxillary right central incisor, and intrusive luxation of maxillary upper left central incisor. **B**, Note severe inflammatory root resorption of maxillary left central incisor and the replacement resorption of maxillary right incisor. Endodontic therapy with calcium hydroxide was initiated for the maxillary right lateral incisor. Splint was applied 6 months posttrauma because of severe mobility secondary to root resorption.



A



B



C

**Figure 1-3** Fourteen-year-old male sustained avulsion of both maxillary central incisors while playing in a swimming pool. He presented to dental office 2 years after trauma. **A**, Note the infraocclusion of both maxillary central incisors. **B**, Pre-operative radiograph reveals replacement resorption of roots from both teeth. Note the remnants of gutta-percha apically. **C**, Extracted teeth.



## DEFINITION AND CLASSIFICATION OF INJURIES

There are basically two types of injuries to the dentition:

- **Hard tissue injuries:** involving the teeth, alveolar bone, and other facial bones (see Chapters 3 to 7).
- **Soft tissue injuries:** involving the facial skin, lips, mucosa (cheeks and periodontium), soft tissues of the hard and soft palate, and tongue (see Chapter 8).

Since so many different types of injuries can occur to the dentition, the various classifications are defined below and elaborated upon in the subsequent chapters.

### HARD TISSUE INJURIES

#### Tooth Injuries

##### Crown fractures

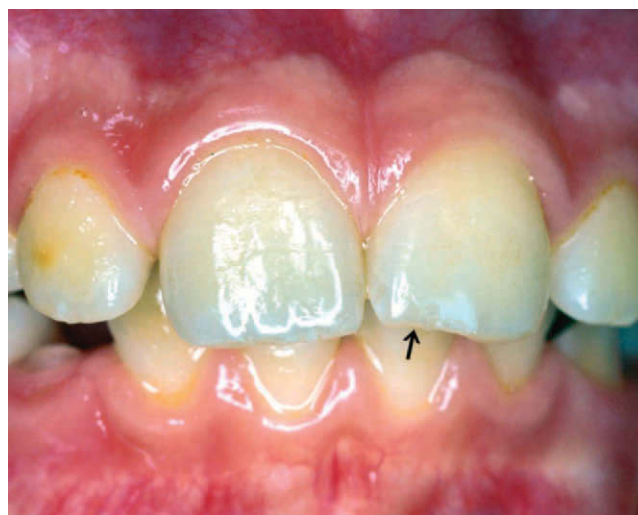
Crown fractures are the most common type of dental trauma.<sup>5,17,43</sup> The type of injury that may occur depends on the age of the patient and the severity and direction of the trauma. These injuries are described in great detail in Chapter 3, and are summarized below:

- **Enamel infraction:** presents as a crack or *craze line* in the enamel. It is usually hairline thin in appearance and is often only noticeable when light is transilluminated through the crown. There is a tendency for these cracks to retain stains, which may create cosmetic concerns (Fig. 1-4).
- **Uncomplicated crown fracture:** this is a fracture of the crown that involves only the enamel or the enamel and dentin, with *no* pulp exposure (Fig. 1-5).
- **Complicated crown fracture:** this is a fracture of the crown that involves enamel and dentin, which is deep enough to result in a *pulp exposure* (Fig. 1-6).

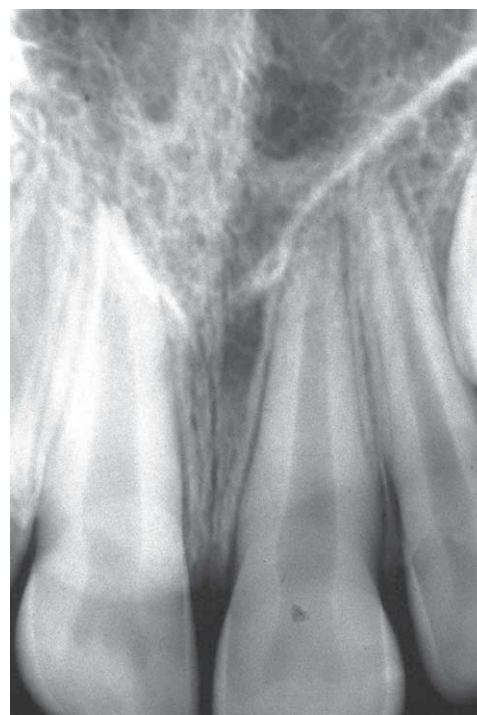
##### Root fractures

Occasionally, there may be an injury of the tooth that does not directly affect the crown of the tooth, but rather causes a fracture through the root. This fracture may be vertical, horizontal, or oblique in relationship to the long axis of the root. These injuries are elaborated on in Chapter 4, and are summarized below:

- **Crown-root fracture:** this fracture involves both the crown and the root at the same time. Typically there is a horizontal or oblique cervical fracture, which extends just below the attachment apparatus or into the alveolar bone. Often the crown is separated completely from the root; in some cases, it is held in place only by the attachment apparatus (Fig. 1-7).
- **Intraalveolar root fractures:** these injuries involve a fracture of the root that is completely encased within bone. The fracture may be horizontal (also called *transverse*) or more diagonal (also called *oblique*), and typically divides the root into two *fragments*: a *coronal* fragment and an *apical* fragment (Fig. 1-8).



A



B

**Figure 1-4** Eleven-year-old male with concussion trauma to both maxillary central incisors. **A**, Enamel fracture of maxillary left central incisor (*arrow*). **B**, Preoperative radiograph appears within normal limits.

#### Luxation injuries

When a traumatic injury to a tooth seems to cause its displacement from the socket, it is termed a *luxation* injury. The *type* of luxation injury relates to the direction and severity of the injury. Although elaborated in great detail in Chapters 5 and 6, the luxation categories are summarized below:

- **Concussion:** when the tooth is traumatized by an impact, but does not change from its normal position.



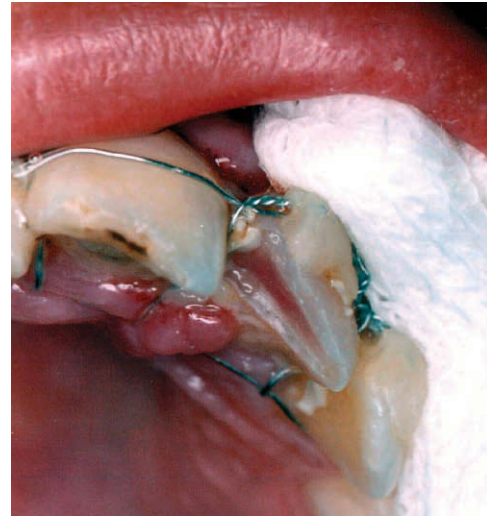
A



B

**Figure 1-5** Eight-year-old female fell and hit her face. **A**, Uncomplicated crown fractures of both maxillary central incisors. **B**, Radiograph reveals immature apices.

- **Subluxation:** when the tooth sustains an impact that causes slight mobility with no significant displacement from its socket (Fig. 1-9).
- **Lateral luxation:** implies that the tooth has been displaced within its socket in a buccal-lingual or labial-palatal direction.
- **Intrusion:** when the tooth is displaced in an apical direction within the alveolus.
- **Extrusive luxation:** when the tooth is displaced from its socket in a coronal direction.
- **Avulsion (or exarticulation):** when the tooth has been completely dislodged out of its alveolar socket (Fig. 1-10).



**Figure 1-6** Eight-year-old female fell and hit her mouth, resulting in complicated crown fractures (exposed pulps) of both maxillary central incisors. Note pulp hyperplasia in the maxillary left central incisor.

### Alveolar Injuries

There are several types of fractures that can occur to the bone secondary to dental injuries. *Comminuted* fractures are multiple small fractures of the alveolar socket that can typically arise from luxation injuries. Likewise, there can be lateral, facial, or lingual fractures of the alveolar socket. In more severe injuries, there may also be fractures of the alveolar bone with or without any involvement of any tooth socket. The classification, assessment, and management of these fractures are described in Chapter 7.

### SOFT TISSUE INJURIES

Concomitant with most dental injuries is trauma to the surrounding soft tissues, including the facial skin, lips, oral mucosa, gingiva, frenum, hard and soft palate, and the tongue. Recognition and management of these injuries is imperative and is detailed extensively in Chapter 8.

## ETIOLOGY AND EPIDEMIOLOGY

Many studies have investigated the etiology and epidemiology of dental trauma.<sup>5,8,39,41</sup> They report on the type, location, prevalence, and cause of the injuries. However, there is a certain amount of variability between the quantitative findings of many of these studies. This is not surprising since many of the studies have gathered information from vastly different subpopulations, with varying factors such as environmental, geographic, climatic, and socioeconomic conditions.<sup>5,36</sup> For example, retrospective data gathered from a hospital-based dental treatment center might reveal facial



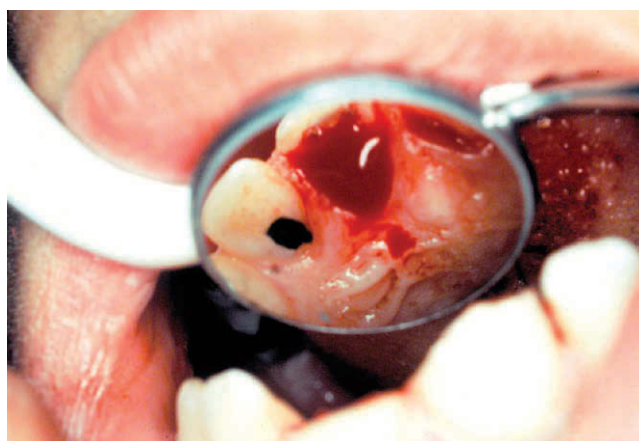
A



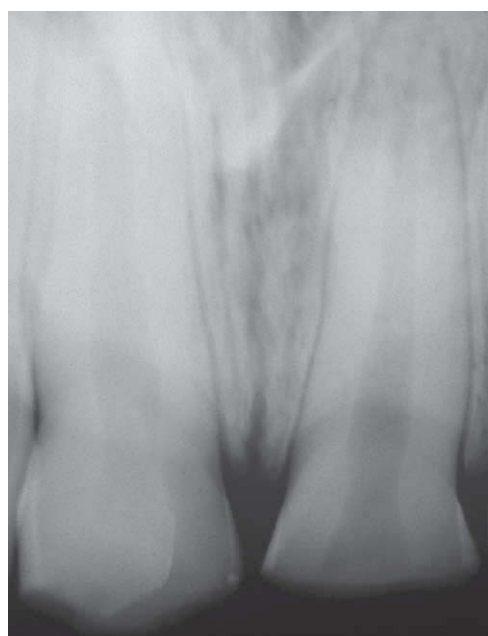
B



C



D



E

**Figure 1-7** Twenty-five-year-old male was seen subsequent to an automobile crash. **A**, Crown-root fractures of both maxillary central incisors. **B**, Palatal view. **C**, Preoperative radiograph revealing fracture lines. **D**, Immediately after coronal fragment removed from maxillary left central incisor. **E**, Posttreatment radiograph revealing extent of the pulp exposures.





**Figure 1-8** Radiograph showing intraalveolar root fractures of both maxillary central incisors.

injuries with a greater severity than those injuries that are reported by a dental clinic outside of a hospital.<sup>29</sup> Since various subpopulations may give conflicting data as to when, where, and how various traumatic injuries typically occur, an evaluation of the many retrospective and prospective studies can only give us general *trends* and *predetermining factors*.

## TRENDS

### Cause of Injury

The most common cause of dental injuries is falls, comprising between 26% and 82% of all sustained injuries, depending on the subpopulation investigated.<sup>1,7,37,39,51</sup>

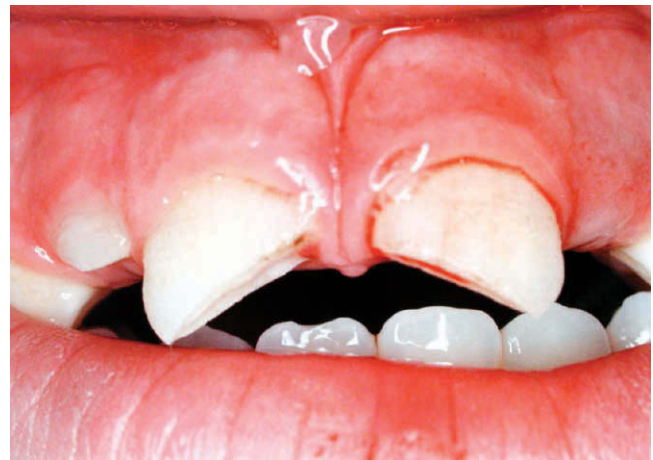
Sports-related injuries are generally the second most common cause of dental injuries.<sup>14,34,38</sup>

### Occurrence of Injury

Because of where young children tend to spend most of their time, it is not surprising that injuries to primary teeth tend



**A**

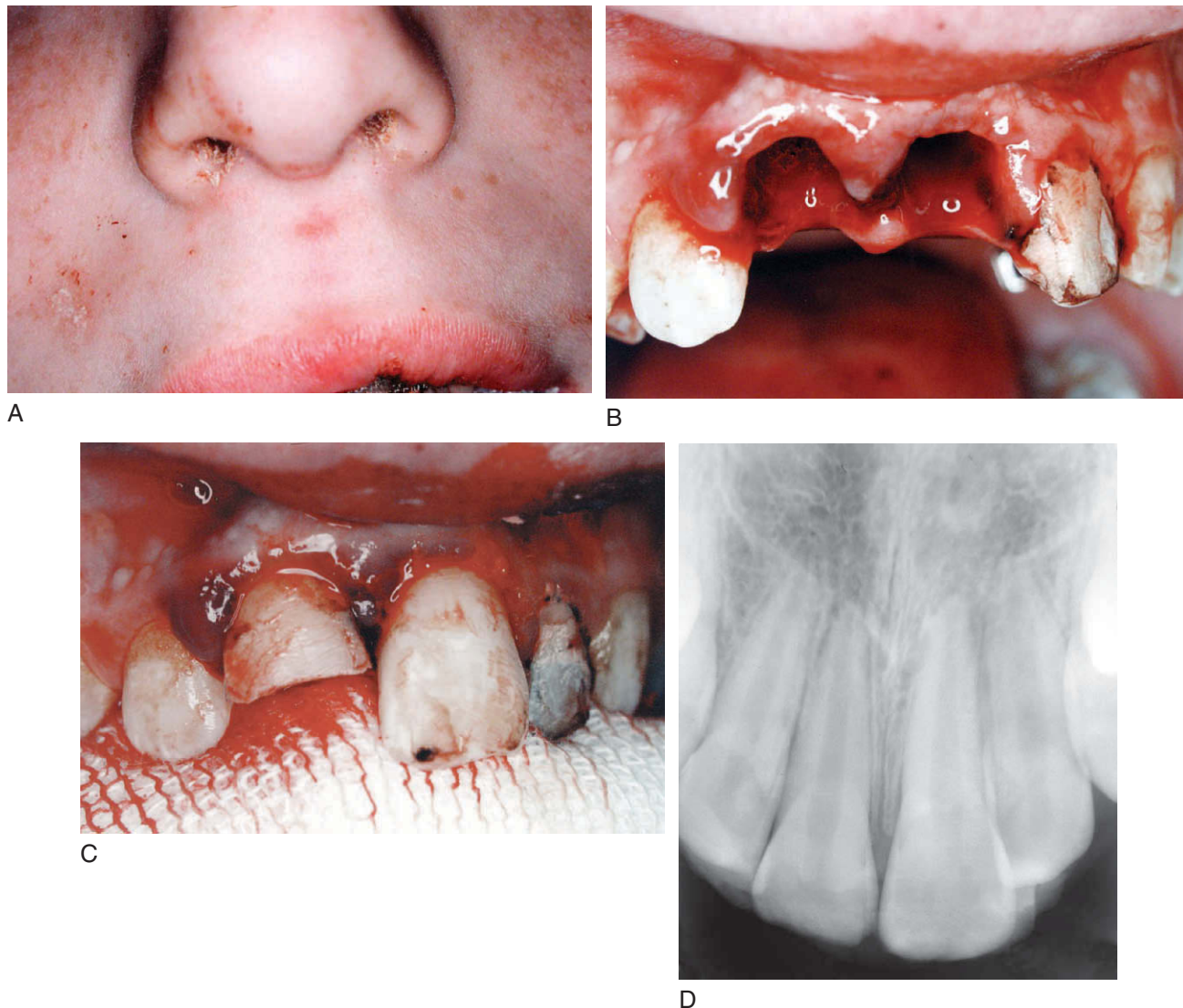


**B**



**C**

**Figure 1-9** Seven-year-old male fell on his face while playing, came to dental office 1 day after the trauma. Note sulcular bleeding of maxillary left central incisor as a result of subluxation. **A** and **B**, Both maxillary central incisors presented with uncomplicated crown fractures (no pulp exposures). The blood at the site of the crown fracture (maxillary left central incisor) was spread from the sulcular bleeding. **C**, Pretreatment radiograph reveals immature apices.



**Figure 1-10** Eleven-year-old male fell while running, causing avulsion of both maxillary central incisors. He presented to the dental office 1 hour after trauma with the teeth soaking in a water container. **A**, Note the facial lacerations. **B**, Copious alveolar and gingival bleeding. **C**, Both incisors were immediately replanted; patient is biting on gauze while the splint is being fabricated. **D**, Post-treatment radiograph after replantation.

to happen more at home, whereas injuries to permanent teeth tend to occur most often outside the home.<sup>18,32,39</sup> These injuries tend to be from bicycle falls, motor vehicle crashes, and fights.<sup>15,29,48</sup>

### Gender Significance in Injuries

For older children, boys tend to have more dental injuries than girls.<sup>1,26,27,35,36</sup> These findings could be a result of typically more aggressive play from boys and an earlier introduction to competitive sports. However, in younger children, there is not much of a difference in the frequency and severity of dental injuries between the sexes. This is not surprising because at a younger age, the type of play is very similar between boys and girls.<sup>12</sup>

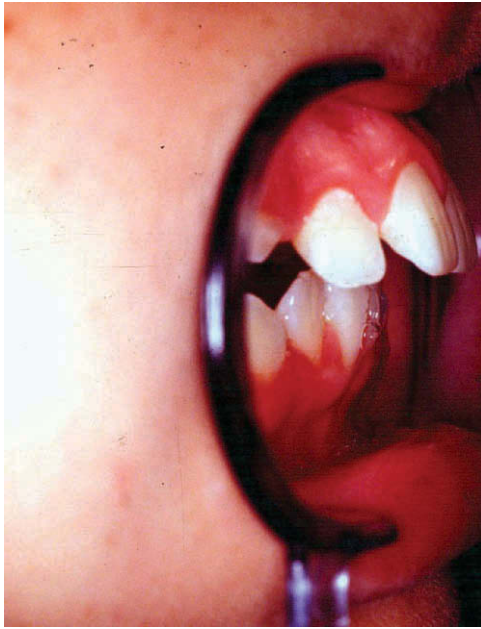
### Type of Injury

For permanent teeth, *uncomplicated crown fractures* tend to be the most common type of dental injury.<sup>2,24,39</sup> The types of injuries of highest frequency for primary teeth tend to be *luxations*.<sup>19,24,39</sup> This is not such a surprising finding since primary teeth are rooted in more resilient and elastic supporting structures. When an injury is sustained to these teeth, there is a tendency for these teeth to become displaced rather than fractured.<sup>3,39</sup>

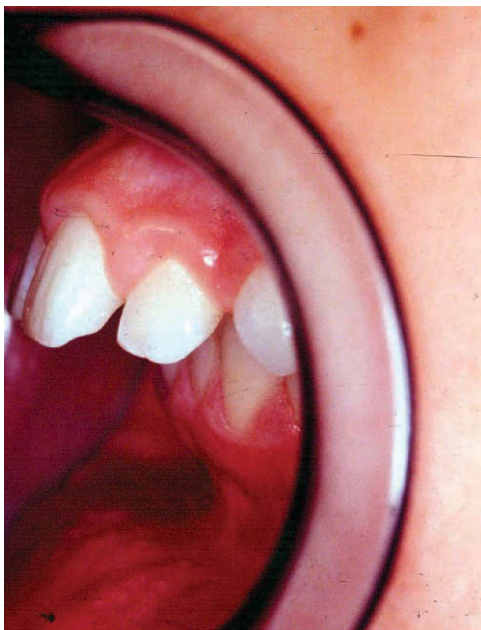
### Dental Location of Injury

Epidemiological studies have shown that the most common tooth to become traumatized is the maxillary central incisor followed by the maxillary lateral incisor.<sup>5,8,51</sup>





A



B

**Figure 1-11** Twelve-year-old male was seen with protrusion of maxillary central incisors. **A**, Right profile. **B**, Left profile.

## PREDETERMINING FACTORS

Because most dental injuries occur to maxillary central incisors, it is understandable that *malocclusion* and the lack of *natural protection* to these teeth will predispose them to injury. Severe overjet of the upper central incisors (i.e., overjet greater than 3 mm) will predispose these teeth to up to five times the risk of trauma compared with a normal overjet.<sup>6,40,44</sup> The natural protection for these teeth involves the adequate coverage by the patient's upper lip. It has been



A



B

**Figure 1-12** Nine-year-old female was seen with oral breathing. **A**, Incompetent, hypotonic lips. **B**, Parafunctional habits including atypical swallowing with interposition of the tongue between maxillary and mandibular teeth.

shown that incompetent lip coverage tends to precipitate more severe injuries to the teeth<sup>9,11,22</sup> (Figs. 1-11 and 1-12).

*Socioeconomic background* may also have some impact. Typically there are more severe injuries in children of a lower socioeconomic subpopulation.<sup>28,31</sup> This may have to do with inadequate supervision, an increase in assault frequency, and/or the lack of adequate patient education pertaining to prevention.

*Orthodontic appliances* may create more soft tissue injuries when trauma presents, potentially causing increased injury to the gingiva and lips.<sup>46</sup>

*Acute medical problems*, such as seizures, heart attack, or stroke, may predispose the patient to falling and traumatically injuring the dentition.<sup>10,23</sup>

*General anesthetic* may also predispose the patient to intraoral trauma from the placement of an endotracheal



tube. It has been reported that dental trauma may occur in as many as 18% of patients who are intubated.<sup>20,33,42</sup>

## PREVENTION

Traumatic injuries to the dentition are difficult to anticipate. However, the exercising of good judgment and prevention is the best defense. Helmets, seat belts, and properly constructed mouthguards have had a tremendous impact on diminishing the severity of dental injuries. For example, it has been shown that bicycle helmets have reduced the incidence of facial trauma by more than 60%.<sup>25,34,45,49</sup> Chapter 11 elaborates on other areas pertaining to the protection and prevention of dental trauma.

## CONCLUSION

Each of the above topics and more is described in great detail in the subsequent chapters. It is the editors' and authors' intention that with this information the clinician will be able to efficiently assess a traumatic injury to the dentition and use the treatment plan that will most effectively result in the best prognosis for the patient.

## REFERENCES

- Al-Jundi SH: Dental emergencies presenting to a dental teaching hospital due to complications from traumatic dental injuries, *Dent Traumatol* 18:181-185, 2002.
- Al-Jundi SH: Type of treatment, prognosis, and estimation of time spent to manage dental trauma in late presentation cases at a dental teaching hospital: a longitudinal and retrospective study, *Dent Traumatol* 20:1-5, 2004.
- Andreasen JO, Andreasen FM, editors: Textbook and color atlas of traumatic injuries to the teeth, ed 3, St Louis, 1994, Mosby.
- Bakland LK: Root resorption, *Dent Clin North Am* 36:491-505, 1992.
- Bastone EB, Freer TJ, McNamara JR: Epidemiology of dental trauma: a review of the literature, *Australian Dent J* 45:1-8, 2000.
- Bauss O, Rohling J, Schweska-Polly R: Prevalence of traumatic injuries to the permanent incisors in candidates for orthodontic treatment, *Dent Traumatol* 20:61-66, 2004.
- Bishai D, McCauley J, Trifiletti LB et al: The burden of injury in preschool children in an urban medical aid managed care organization, *Ambul Pediatr* 2:279-283, 2002.
- Borssen E, Holm AK: Treatment of traumatic dental injuries in a cohort of 16-year-olds in northern Sweden, *Endod Dent Traumatol* 16:276-281, 2000.
- Brin I, Ben-Bassat Y, Heling I et al: Profile of an orthodontic patient at risk of dental trauma, *Endod Dent Traumatol* 16:111-115, 2000.
- Buck D, Baker GA, Jacoby A et al: Patients' experiences of injury as a result of epilepsy, *Epilepsy* 38:439-444, 1997.
- Burden DJ: An investigation of the association between overjet size, lip coverage, and traumatic injury to maxillary incisors, *Eur J Orthod* 17:513-517, 1995.
- Caldas AF Jr, Burgos MEA: A retrospective study of traumatic dental injuries in a Brazilian dental trauma clinic, *Dent Traumatol* 17:250-253, 2001.
- Cohen S, Blanco L, Berman L: Early radiographic diagnosis of inflammatory root resorption, *Gen Dent* 3:235-240, 2003.
- Cornwell H: Dental trauma due to sport in the pediatric patient, *J Calif Dent Assoc* 33:457-461, 2005.
- Da Silva AC, Passeri LA, Mazzonetto R et al: Incidence of dental trauma associated with facial trauma in Brazil: a 1-year evaluation, *Dent Traumatol* 20:6-11, 2004.
- Davis RM: BMJ bans "accidents." Accidents are not unpredictable, *BMJ* 322:1320-1321, 2001.
- Davis GT, Knott SC: Dental trauma in Australia, *Aust Dent J* 29:217-221, 1984.
- Dorney B: Inappropriate treatment of traumatic dental injuries, *Aust Endod J* 25:76-78, 1999.
- Flores MT: Traumatic injuries in the primary dentition, *Dent Traumatol* 19:118, 2003.
- Fung BK, Chan MY: Incidence of oral tissue trauma after the administration of general anesthesia, *Acta Anaesthesiol Sin* 39:163-167, 2001.
- Kahabuka FK, Ntabaye MK, van't Hof MA et al: Effect of a consensus statement on initial treatment choice for traumatic dental injuries, *Dent Traumatol* 17:159-162, 2001.
- Kania MJ, Keeling SD, McGorray SP et al: Risk factors associated with incisor injury in elementary school children, *Angle Orthod* 66:423-432, 1996.
- Karasu HA, Uyanik LO, Kocyigit ID: Displacement of avulsed tooth into soft tissue of chin resulting from epileptic attack trauma, *NY State Dent J* 71:26-28, 2005.
- Kargul B, Caglar E, Tanboga I et al: School dental trauma in Turkish children, Istanbul, *Dent Traumatol* 19:72-75, 2003.
- Kubiak R, Slongo T: Unpowered scooter injuries in children, *Acta Paediatr* 92:50-54, 2003.
- Liew VP, Daly CG: Anterior dental trauma treated after-hours in Newcastle Australia, *Community Dent Oral Epidemiol* 14:362-366, 1986.
- Malikaew P, Watt RG, Sheiham A: Associations between school environments and childhood traumatic dental injuries, *Oral Health Prev Dent* 1:255-266, 2003.
- Marcenes W, Murray S: Social deprivation and traumatic dental injuries among 14-year-old schoolchildren in Newham, London, *Dent Traumatol* 17:17-21, 2001.
- Martin IG, Daly CG, Liew VP: After-hours treatment of anterior dental trauma in Newcastle and western Sydney: a four-year study, *Aust Dent J* 35:27-31, 1990.
- Neira J, Bosque L: The word accident: no chance, no error, no destiny, *Prehospital Disaster Med* 19:188-189, 2004.
- Nicolau B, Marcenes W, Sheiham A: The relationship between traumatic dental injuries and adolescents' development along the life course, *Community Dent Oral Epidemiol* 31:306-313, 2003.
- Onetto JE, Flores MT, Garbarino ML: Dental trauma in children and adolescents in Valparaíso, Chile, *Endod Dent Traumatol* 10:223-227, 1994.
- Owen H, Waddell-Smith I: Dental trauma associated with anaesthesia, *Anaesth Intensive Care* 28:133-145, 2000.
- Padilla R, Felsenfeld AL: Treatment and prevention of alveolar fractures and related injuries, *J Craniomaxillofac Trauma* Fall 3:22-27, 1997.
- Perez R, Berkowitz R, McIlveen L et al: Dental trauma in children survey, *Endod Dent Traumatol* 7:212-213, 1991.
- Perheentupa U, Laukkanen P, Veijola J et al: Increased lifetime prevalence of dental trauma is associated with previous non-dental injuries, mental distress and high alcohol consumption, *Dent Traumatol* 17:10-16, 2001.

37. Powell EC, Jovtis E, Tanz RR: Incidence and description of stroller-related injuries to children, *Pediatrics* 110(5):e62, 2002.
38. Ranalli DN, Demas PN: Orofacial injuries from sport: preventive measures for sports medicine, *Sports Med* 32:409-418, 2002.
39. Rocha MJ, Cardoso M: Traumatized permanent teeth in Brazilian children assisted at the Federal University of Santa Catarina, Brazil, *Dent Traumatol* 17:245-249, 2001.
40. Shulman JD, Peterson J: The association between incisor trauma and occlusal characteristics in individuals 8-50 years of age, *Dent Traumatol* 20:67-74, 2004.
41. Skaare AB, Jacobsen I: Dental injuries in Norwegians aged 7-18 years, *Dent Traumatol* 19:67-71, 2003.
42. Skeie A, Schwartz O: Traumatic injuries of the teeth in connection with general anesthesia and the effect of use of mouthguards, *Endod Dent Traumatol* 15:33-36, 1999.
43. Stockwell AJ: Incidence of dental trauma in the Western Australian School Dental Service, *Community Dent Oral Epidemiol* 16:294-298, 1998.
44. Stokes AN, Loh T, Teo CS et al: Relation between incisal overjet and traumatic injury: a case control study, *Endod Dent Traumatol* 11:2-5, 1995.
45. Thompson DC, Nunn ME, Thompson RS et al: Effectiveness of bicycle safety helmets in preventing serious facial injury, *JAMA* 276:1974-1975, 1996.
46. Travess H, Roberts-Harry D, Sandy J: Orthodontics. Part 6: Risks in orthodontic treatment, *Br Dent J* 24:196:71-77, 2004.
47. Trope M: Luxation injuries and external root resorption—etiology, treatment, and prognosis, *J Calif Dent Assoc* 28:860-866, 2000.
48. Wood EB, Freer TJ: A survey of dental and oral trauma in south-east Queensland during 1998, *Aust Dent J* 47:142-146, 2002.
49. Woodmansey KF: Athletic mouth guards prevent orofacial injuries: a review, *Gen Dent* 47:64-69, 1999.
50. Zerfowski M, Bremerich A: Facial trauma in children and adolescents, *Clin Oral Investig* 2:120-124, 1998.
51. Zuhail K, Semra OE, Huseyin K: Traumatic injuries of the permanent incisors in children in southern Turkey: a retrospective study, *Dent Traumatol* 21:20-25, 2005.

# PATIENT ASSESSMENT



ZVI FUSS, SHAUL LIN, AND IGOR TESIS

## CHAPTER OUTLINE

### MEDICAL EMERGENCY CONSIDERATIONS

### ORAL EMERGENCY CONSIDERATIONS

#### Patient History

#### Soft Tissue Clinical Examination

##### Extraoral Examination

##### Intraoral Examination

#### Soft Tissue Radiographic Examination

#### Hard Tissue Clinical Examination

##### Alveolar Bone

##### Teeth

##### Observation

##### Mobility

##### Percussion

##### Pulp Vitality

##### Laser Doppler Flowmetry

#### Hard Tissue Radiographic Examination

##### Teeth

##### Alveolar Bone

### PEDIATRIC CONSIDERATIONS

### GERIATRIC CONSIDERATIONS

### CONCLUSION

Traumatic dental injuries are typically emergencies that the clinician must be able to rapidly assess and appropriately manage.<sup>43,46,53,79</sup> The prognosis for each case depends on how immediately and accurately the patient's injuries are diagnosed and treated.<sup>27,78</sup> The importance of proper documentation cannot be overemphasized, not just for medico-legal considerations (see Chapter 9), but also as a detailed reference regarding the traumatic injury. Any complications that may arise in the future will be better managed when accurate documentation is detailed during patient assessment of the traumatic injury.

## MEDICAL EMERGENCY CONSIDERATIONS

The medical status of the patient plays a vital role in the comprehensive management of traumatic injuries. The patient may have been medically compromised before the injury or as a result of the injury. Not only will these medical issues have an impact on the overall dental treatment of the patient, but also a compromised medical status can be life threatening when proper assessment and referral are not made in a timely manner. Therefore a medical evaluation is imperative before any initial dental treatment. Usually a thorough evaluation is made by a physician. However, the clinician should be aware of the general medical issues that may affect emergency dental care.

A thorough medical history should be taken as soon as possible. Details regarding systemic diseases, allergies, and recent hospitalizations are good screening questions.<sup>6,11</sup>

Essential vital signs, such as blood pressure, pulse, and respiration, are important preliminary evaluations. Immediately confirm unimpeded breathing and circulation. Pulse rate and blood pressure should be recorded. Cool, pale skin, perspiration, hypotension, tachycardia, and mental status changes are reliable indicators of shock. In the traumatized patient, shock is due to hypovolemia from hemorrhage in most cases.<sup>83</sup> The incidence of severe hemorrhage





**Figure 2-1** Periorbital hematoma and frontal laceration following a bicycle fall.

secondary to facial fractures is rare; however, it can be life threatening.<sup>14</sup> Decreased pulse rate along with hypertension may indicate a rise in intracranial pressure.<sup>80,86</sup> Gross physical injuries and facial asymmetry should also be noted, which could help in the detection of alveolar and orbital fractures (Fig. 2-1).

Following a traumatic incident, removable prostheses, avulsed teeth, and tooth fragments may become aspirated and cause partial or complete airway obstruction.\* Close observation of the patient is the best way to diagnose an aspirated foreign body.<sup>26</sup> Common findings of foreign body aspiration are coughing, cyanosis, dyspnea, and fever.<sup>54</sup> Any patient suspected of having partial airway obstruction should have chest radiographs taken as quickly as possible to rule out a foreign body within the lungs.<sup>26,44</sup> Abdominal radiographs should also be considered when teeth are missing during the clinical examination.<sup>61</sup>

On presentation, a patient's clinical status is grossly assessed with the Glasgow Coma Scale (GCS) (Table 2-1) to determine the presence and extent of traumatic brain injury.<sup>84</sup> Numerical values for eye opening, motor responses, and verbal responses that indicate the level of consciousness and degree of dysfunction are evaluated. Scores range from 3 to 15; lower scores indicate more severe brain injury.<sup>39,84</sup> With regards to the posttrauma evaluation, questions pertaining to loss of consciousness, dizziness, headache, nausea, and vomiting should be discussed since their presence could indicate possible intracranial injury requiring immediate medical attention.<sup>28,56,65,66</sup>

The aim of a cursory neurological examination is for the clinician to quickly recognize signs of a potential neurological crisis and to determine whether the traumatized patient should be referred to a physician for emergency care. Failure to recognize a medically compromised state may precipitate intracranial hypertension, systemic hypotension, hypoxemia, hypercapnia, or infection.<sup>20,28,56,65,66</sup> Difficulty with communication, unusual motor activity, signs of disorientation, loss of consciousness, seizures, headache,

**TABLE 2-1**

**GLASGOW COMA SCALE  
FOR EVALUATION OF  
THE PRESENCE AND  
EXTENT OF TRAUMATIC  
BRAIN INJURY**

|                       |                         |   |
|-----------------------|-------------------------|---|
| Eye opening           | Spontaneous             | 4 |
|                       | To verbal command       | 3 |
|                       | To pain                 | 2 |
|                       | None                    | 1 |
| Verbal responsiveness | Oriented                | 5 |
|                       | Confused                | 4 |
|                       | Inappropriate words     | 3 |
|                       | Incomprehensible sounds | 2 |
|                       | None                    | 1 |
| Motor response        | Obeys                   | 6 |
|                       | Localizes               | 5 |
|                       | Withdraws (pain)        | 4 |
|                       | Flexion (pain)          | 3 |
|                       | Extension (pain)        | 2 |
|                       | None                    | 1 |
| Total: _____          |                         |   |

Lower scores indicate more severe brain injury.

amnesia, and nausea or vomiting could indicate intracranial injury, which will require immediate referral for emergency medical treatment.<sup>39,59</sup>

Clear cerebrospinal fluid through the nose (rhinorrhea) or the ear (otorrhea) may indicate fractures of craniofacial osseous structures. Specifically the patient may have disruption of the anterior cranial base, most commonly at the cribriform plate of the ethmoid bone associated with nasoorbitoethmoid fractures, or disruption of the posterior wall of the frontal sinus.<sup>29</sup>

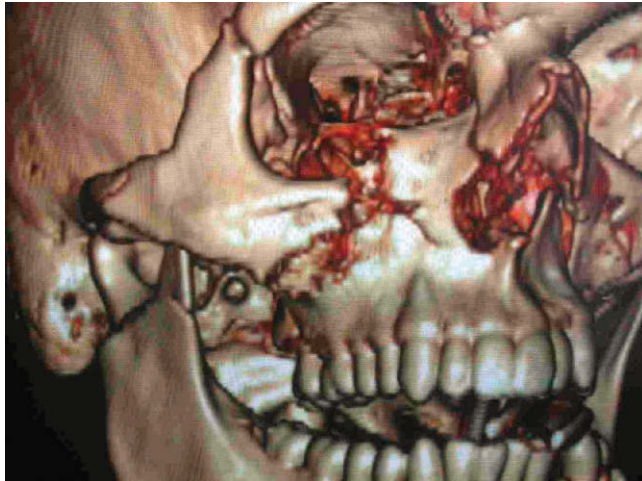
The inability of patients to open their eyes may also indicate an underlying neurological injury.<sup>39</sup> Diplopia (double vision), caused by extraocular muscle imbalance, is a common complication of fractures of the zygomatic-maxillary complex (Fig. 2-2),<sup>91</sup> with blow-out fractures of the orbit as the most common cause.<sup>42</sup> The presence of monocular diplopia (double vision of one eye due to detached retina or lens dislocation) could indicate injury to the globe.<sup>1</sup> Both pupils should be equal in size and reactive to light, otherwise underlying brain injury should be suspected.<sup>77</sup>

Discomfort or paresthesia of the extremities during head movement may indicate cervical vertebral fractures. When vertebral injury is suspected, the patient should not be moved and medical referral should be made immediately.<sup>20,38</sup>

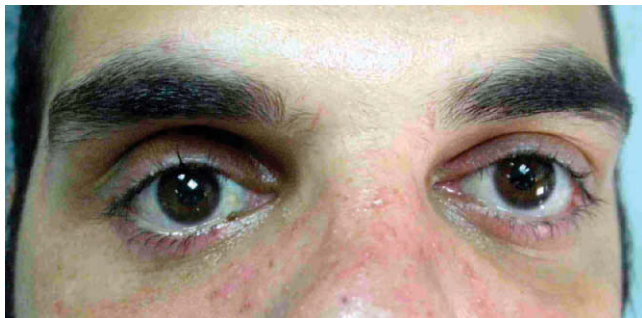
Inability of the patient to protrude the tongue suggests possible damage to the hypoglossal nerve. The patient's ability to hear and maintain a postural balance will confirm normal auditory and vestibular function.<sup>20</sup>

The presence of localized areas of anesthesia or paresthesia on the patient's face, although not necessarily a medical

\*References 24, 26, 44, 50, 71, 82.



A



B

**Figure 2-2** A, Zygomatic complex fracture visualized by 3-D CT reconstruction. B, Right exophthalmos due to zygomatic complex fracture.

emergency, could indicate damage to the trigeminal nerve with concomitant alveolar bone fractures<sup>29</sup> (Table 2-2).

Once confirmed that there are no major medical issues, the patient can be evaluated for injuries to the dentition.

## ORAL EMERGENCY CONSIDERATIONS

### PATIENT HISTORY

Assessment of the traumatized patient begins with a careful history regarding *where*, *when*, and *how* the trauma occurred. *Where* the trauma occurred (location) can provide useful information as to the degree of bacterial contamination and the need for tetanus prophylaxis. For a clean wound, a tetanus booster is necessary if the last one was more than 10 years ago; for contaminated wounds, if more than 5 years ago (Fig. 2-3).<sup>76,83</sup>

*When* the injury occurred is also important to determine the necessary treatment and to assess the future prognosis of treatment.<sup>27</sup> Time between the trauma and immediate treatment plays an important role in dental avulsion and

**TABLE 2-2**

**SUMMARY OF NEUROLOGICAL EVALUATION OF THE TRAUMATIZED PATIENT AND POSSIBLE CRANIAL NERVE INJURIES**

| CLINICAL SIGNS                                  | POTENTIAL DAMAGE TO CRANIAL NERVE |
|-------------------------------------------------|-----------------------------------|
| Loss of the sense of smell                      | Olfactory nerve (I)               |
| Apparent loss of the sense of taste             |                                   |
| Diplopia (double vision)                        | Optic nerve (II)                  |
| Blurred vision                                  | Oculomotor nerve (III)            |
| Pupils do not react to light                    | Trochlear nerve (IV)              |
| Pupils are not equal in size                    | Abducens nerve (VI)               |
| Areas of paresthesia on the face and upper neck | Trigeminal (V)                    |
| Loss of sensation on anterior tongue            |                                   |
| Inability to wrinkle forehead and nose          | Facial nerve (VII)                |
| Inability to squeeze the eyes                   |                                   |
| Inability to elevate upper lip                  |                                   |
| Impaired hearing                                | Vestibulocochlear nerve (VIII)    |
| Inability to maintain postural balance          |                                   |
| Difficulty in swallowing                        | Glossopharyngeal (IX)             |
| Abnormal speech with hoarse voice               | Vagus (X)                         |
| Inability to protrude the tongue                | Hypoglossal (XII)                 |
| Ipsilateral tongue deviation                    |                                   |

luxation cases. As discussed in Chapters 5 and 6, delayed treatment of a luxated or avulsed tooth makes placement or manipulation of the tooth back to its original position difficult. The overall prognosis is decreased by situations that increase the potential for various types of root resorption.<sup>7,33</sup>

As discussed in Chapter 3, a longer period between a traumatic pulp exposure and treatment may result in an increased possibility of pulp necrosis, thus compromising cases of teeth with immature root development.<sup>8</sup>

Clues for identifying other related injuries may be provided by questioning the patient on *how* the traumatic injury occurred. For example, trauma to the cheek may cause a zygomatic fracture, temporomandibular joint (TMJ) disorder, or possible tooth fractures in this area.<sup>11</sup> As discussed in Chapter 7, falling, either as the cause or the result of the primary injury, may cause maxillary and/or mandibular alveolar bone fractures or injury to other proximal areas of the dentition. The pattern of tooth injury depends on the site, direction, and resilience of the periodontal structures surrounding the tooth.<sup>25</sup>



A



B

**Figure 2-3** A and B, Facial soft tissue trauma secondary to fall in street.

The clinician must also be aware of intentional injuries. Abused children or victims of domestic violence have resulted in an increased incidence of facial, head, or neck injuries.<sup>25,88</sup> Often these injuries are first detected by visible soft tissue trauma, as described in Chapter 8.



**Figure 2-4** Twelve-year-old girl with mandibular angle fracture and extensive swelling and bruising of the overlying soft tissue.

## SOFT TISSUE CLINICAL EXAMINATION

### Extraoral Examination

Lacerations, abrasions, and contusions of facial soft tissues are generally associated with trauma and are readily seen on external clinical examination. Where soft tissue injury is observed, the area should be washed gently with mild detergent, and the location of any bleeding noted. When hemorrhage is present, finger pressure should be applied to the site for several minutes until excessive bleeding is stopped. For extensive hemorrhage, a suture may be necessary proximal to the laceration. Asymmetry, flatness of the face, and changes in facial height or width should be noted. These soft tissue changes could indicate underlying skeletal trauma (Fig. 2-4). Ecchymosis seen behind the ear (*Battle's sign*) or bilaterally in the periorbital areas (*raccoon eyes*), concomitant with rhinorrhea or otorrhea, may indicate a basal skull fracture that requires immediate medical attention.<sup>43,73</sup>

### Intraoral Examination

Complete intraoral examination begins with visual observation of the intraoral soft tissues. Signs of lacerations or penetrating injuries should be evaluated. Careful irrigation and suctioning of the intraoral tissues, using warm water of the triple syringe, will allow better viewing of these tissues without provoking pain from potentially injured teeth. When there is major bleeding present in the oral cavity, immediate detection of origin is crucial followed by arresting the bleeding by manual pressure with iced gauze (if available). When digital (manual) pressure is not sufficient,





A



B

**Figure 2-5** Bleeding from the sulcus can indicate displacement injuries from luxation or crown-root fracture.

administering a local anesthetic with a vasoconstrictor (1:50,000) and, if necessary to stop the bleeding, suturing mesial to the bleeding vessel. Unless bleeding is controlled, diagnosis of the extent of the injury will be difficult to assess. Further treatment options for the management of soft tissue injuries are presented in Chapter 8.

Soft tissues of the periodontium should also be examined. Bleeding from the sulcus (Fig. 2-5) may indicate a crown-root fracture (see Chapter 4), tooth displacement (see Chapters 5 and 6), or an alveolar fracture (see Chapter 7).

Palpation of the oral mucosa is also essential. Soft or hard swelling may indicate the presence of an embedded foreign body (Fig. 2-6).

The sublingual area should be examined for signs of ecchymosis suggesting a mandibular fracture. Maxillary fractures may be observed by palpable moveable segments or facial edema.<sup>29</sup>



**Figure 2-6** Laceration and swelling of lip can indicate foreign body embedded in the soft tissue.



**Figure 2-7** Hematoma indicative of underlying alveolar fracture.

## SOFT TISSUE RADIOGRAPHIC EXAMINATION

Radiographs of the soft tissue are not typically considered in a clinical examination. However, in the event of traumatic injury to the dentition, a simple periapical radiograph may help detect embedded tooth fragments or foreign bodies in the soft tissues of the oral cavity, such as the lips and cheek (see Chapter 8).<sup>18,23</sup>

## HARD TISSUE CLINICAL EXAMINATION

### Alveolar Bone

Visual observation may easily detect alveolar bone fractures that protrude through the overlying mucosa. However, visualization for hematoma and gentle palpation may detect covered fractures when the overlying mucosa is not lacerated (Fig. 2-7). Pain, malocclusion, and mobility of fractured segments provide additional signs of alveolar bone fractures.



A



B

**Figure 2-8** **A**, Avulsed tooth and **B**, intruded tooth shows the importance of locating displaced teeth.

As a follow-up to alveolar fractures, pulpal necrosis may be a sequela to alveolar fractures when in proximity to the apical region of the root.<sup>49,67</sup> In areas where avulsions, luxations, or other tooth trauma is detected, the integrity of the proximate alveolar bone should be examined for any fractures. As described in Chapter 7, any suggestion of alveolar fractures should be further investigated with an appropriate radiograph.

## Teeth

### Observation

Clinical evaluation of the teeth subsequent to traumatic injury begins with the search for any missing, displaced, or fractured teeth (Fig. 2-8). For an avulsed tooth, treatment should be initiated immediately. The tooth is replanted according to the guidelines recommended in Chapter 6.

Delay in this treatment will significantly reduce the prognosis of the tooth (Fig. 2-9).

Occasionally a tooth may undergo a change in color weeks, months, or years after a traumatic injury (Fig. 2-10, *A* and *B*). This could be due to pulpal hemorrhage into the dentinal tubules, excessive pulp chamber calcification secondary to the injury, or internal or cervical resorption (Fig. 2-10, *C* and *D*). Color change does not necessarily indicate that the tooth is nonvital. As described later, pulp vitality must be ascertained to assure proper treatment.

When there is evidence of tooth damage, further evaluation should be made to determine whether pulp exposure or enamel cracks are present. In case of pulp exposure, its size and location should be documented (Fig. 2-11, *A* and *B*). Although pulpal inflammation does not progress rapidly after traumatic exposure, approximately 2 mm after 48 hours,<sup>22</sup> treatment should be prompt to prevent bacterial contamination and preserve pulpal vitality,<sup>41</sup> especially when the remaining vital pulp is associated with an immature root (Fig. 2-11, *C*) (see Chapter 3).

Enamel cracks or infractions occur in 10.5% to 12.5% of acute traumatized incisors<sup>75</sup> and may account for various symptoms, especially hypersensitivity to cold or inhaled air. To improve visualization of potential cracks, fiber optic light is helpful via transillumination. The light source is placed just above the gingival sulcus parallel to the tooth surface to illuminate the clinical crown<sup>57</sup> (Fig. 2-12). Enamel cracks can be best observed with magnification, particularly with the dental operating microscope<sup>17</sup> that permits visualization from 4× to 25× magnification.

Symmetry of the observed teeth should be specifically noted, with special attention directed towards any abnormal positioning of any tooth (or teeth), including rotation, malocclusion, dislodgment, infraposition, or extrusive positioning (Fig. 2-13).<sup>11</sup>

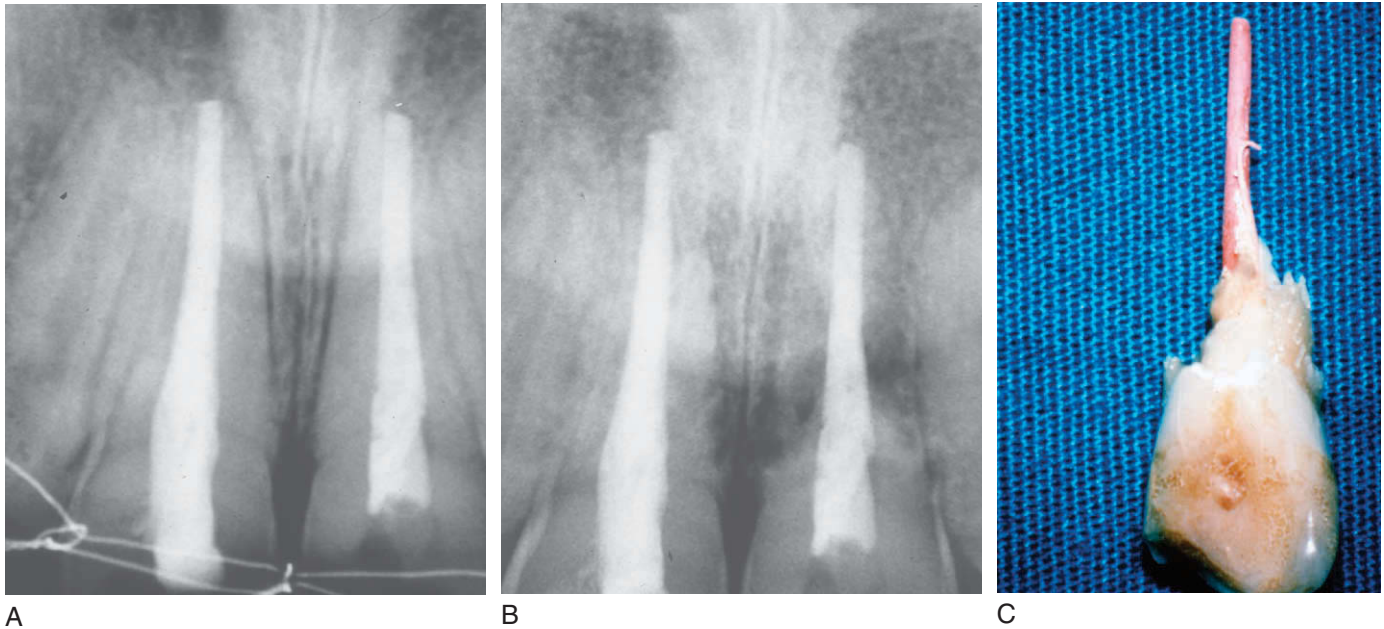
### Mobility

Tooth mobility is observed by objectively moving the tooth, using two instruments (typically the back ends of dental mirror handles), with one instrument on the facial and the other on the palatal of the tooth (Fig. 2-14). An effort is made to move the tooth in all directions; abnormal mobility most often occurs facio-lingually.<sup>16</sup> Degree of mobility is recorded as:<sup>11</sup>

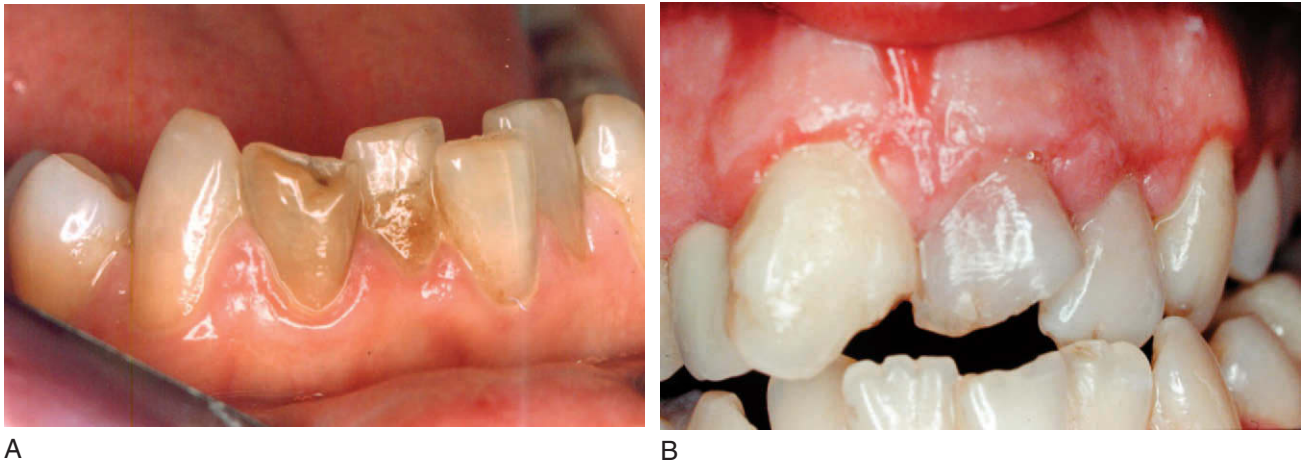
- 0 = no mobility
- +1 = less than 1 mm of horizontal movement
- +2 = more than 1 mm of horizontal movement
- +3 = more than 1 mm of horizontal movement and depressible within the socket

Increased mobility indicates subluxation and lateral luxation, with an associated alveolar bone fracture. The total lack of mobility may indicate the presence of an intrusive luxation. A tooth may become ankylosed (fused to the bone, also known as *replacement resorption*) weeks or months after the trauma (described in greater detail in Chapter 6). When tooth mobility is evaluated, care must be taken to see





**Figure 2-9** A, Avulsed and replanted tooth as seen just after trauma and endodontic treatment and subsequent radiograph 6 months later (B) revealing extensive resorption. C, Following extraction, virtually all that remained was gutta percha.



**Figure 2-10** A, Crown discoloration after trauma may be more yellow or gray (B).

*Continued*

whether the *tooth* moves, or the *surrounding alveolar segment* moves. Extensive tooth mobility could also indicate a root fracture<sup>9,10,58</sup> (described further in Chapter 4). Recently, *Periotest*<sup>TM</sup> (Siemens, Gulden-Medizintechnik, Bensheim, Germany) has been introduced to evaluate tooth mobility (Fig. 2-15).<sup>58</sup> This is an electronic device that provides a non-invasive objective measurement of the reaction of the periodontium to a defined impact load applied to the tooth crown. Two readings, with 15 minute intervals, should be taken to allow the periodontium time to recover.

#### Percussion

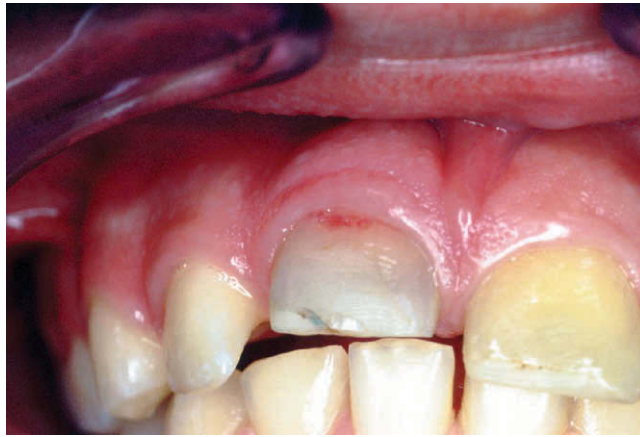
Tooth percussion should initially be performed using a gentle touch with the fingertip, followed by a light percus-

sion with the fingertip. If no pain is elicited, the next test is with a mirror handle, tapping laterally and then vertically on the tooth crown. Sensitivity to percussion is an indication of damage to the periodontal ligament,<sup>19</sup> which could be a sign of an alveolar fracture, a root fracture, or pulpal necrosis with an acute periradicular abscess. Occasionally the audible *sound* of the percussion may be significant. An intruded or ankylosed tooth may have a dull, metallic, higher-pitched sound relative to the adjacent teeth when percussed.<sup>2,15</sup>

#### Pulp vitality

Tooth vitality is determined by the integrity of its vascular supply to the pulp. Vitality tests generally suggest the health and integrity of the intrapulpal sensory nerve fibers. Teeth





C

**Figure 2-10, cont'd** The crown discoloration may originate from cervical resorption as seen clinically (C) and radiographically (D).



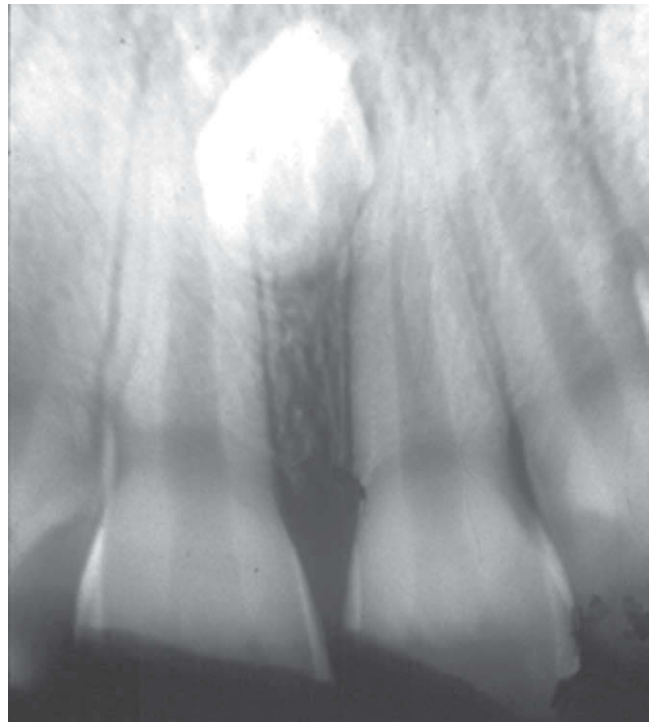
D



A



B



C

**Figure 2-11** A and B, Pulp exposure after complicated crown fracture. C, Radiographic evidence of mature root development.



**Figure 2-12** Transillumination with fiber optic lights can visualize enamel cracks.

with immature root development do not provide a reliable response because the Raschkow plexus is not completely developed. Furthermore, the sensitive A-delta nerve fibers responsible for acute pain and response to vitality tests only mature approximately 4 years after the tooth develops.<sup>40</sup>

Reactions of the dental pulp to traumatic injury can be extremely variable.<sup>30</sup> Although it is not possible to determine the histopathological status of the pulp on the basis of vitality tests alone, there is usually a statistically significant relationship between the lack of response to these tests and pulp necrosis.<sup>45,69,81</sup> When nerve fibers can be stimulated by thermal or electrical means, the pulp vasculature is assumed to be intact.<sup>32</sup> However, the severity of the trauma can have a great impact on whether the pulp survives the injury.

The stage of root development can also be a major factor on how well the pulp will react. Immature root development provides a greater possibility of maintaining pulpal vitality subsequent to the injury, occasionally with a revascularizing pulp.<sup>5,51</sup> In any event, the response of the tooth to pulp vitality tests may provide a false negative response, with an incorrect diagnosis of pulp necrosis. Repetition of pulp vitality tests every 4 to 6 weeks has been suggested to obtain better determination of pulp vitality.<sup>70</sup> It has also been recommended to delay pulp vitality tests for a year or more because the pulp may maintain its vitality, even though pulp testing suggests otherwise.<sup>3,35</sup>

Often, pulp vitality tests can be so subjective that symptoms and radiographic evaluations over time are the only way to objectively determine pulpal vitality. Radiographically, root development should be compared with the adjacent or contralateral tooth, and the periradicular area must be examined to visualize the development of periapical pathosis or resorption.<sup>6,47</sup> The importance of preserving pulpal vitality is to allow normal tooth development and to achieve sufficient dentin wall thickness, thus strengthening the tooth against possible future root fractures.<sup>21</sup>



A



B

**Figure 2-13** A, Clinical evidence of an intruded tooth with radiographic evidence of replacement resorption (B) whereby the root structure is replaced with bone and the loss of the lamina dura.

Thermal and electric pulp tests are generally used to evaluate pulp vitality. The most reliable thermal pulp test is probably the cold test.<sup>32,69</sup> Because ice can melt when applied to the tooth and give potentially false readings, a refrigerant spray (dichlorodifluoromethane), ice water sprayed on a tooth isolated with a rubber dam, and CO<sub>2</sub> snow are more reliable.<sup>32</sup>

Electric pulp tests may not be reliable in young patients (9-13 years old).<sup>32</sup> The electrode should be placed in proximity to the incisal edge.<sup>12,31</sup> Again, this sensitivity test may be of little value immediately following injury.



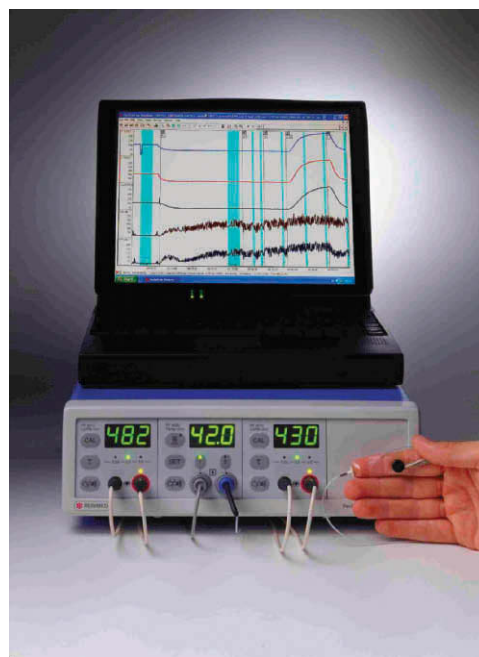
**Figure 2-14** Tooth mobility evaluation using handles of two instruments.



**Figure 2-15** Periotest™ device for the objective evaluation of tooth mobility.

### Laser Doppler flowmetry

Laser Doppler flowmetry (LDF) may be an objective and reliable test of the blood supply to the pulp, enabling the clinician to accurately differentiate between a pulp that is regaining its vitality and one that is becoming necrotic.<sup>62</sup> It uses Doppler shift principles, using a beam of infrared light produced by a laser directed onto and through the tooth crown. The device detects moving red blood cells in the pulp vasculature so as to differentiate vital from nonvital pulp.<sup>36</sup> The LDF has also been used to assess the vitality of traumatized teeth (Fig. 2-16).<sup>35,55,62</sup> However, two different laser Doppler flowmeters were found to detect artificial blood flow in extracted teeth, but the signals representing the



**Figure 2-16** Laser Doppler flowmetry device for the possible detection of pulpal blood flow and vitality.

concentration of moving blood cells were unreliable in both instruments. The information provided by LDF can be ambiguous and should be interpreted with care.<sup>87</sup> LDF is very costly, often rendering it impractical for most dental clinics. *Pulse oximetry*, which measures the blood's oxygen saturation level, may also have some future potential in determining pulp vitality. It has been reported as a possible objective and atraumatic clinical alternative to the present electrical and thermal methods of assessing pulp vitality.<sup>37</sup>

## HARD TISSUE RADIOGRAPHIC EXAMINATION

### Teeth

Periapical and/or occlusal radiographs should be taken to evaluate injured teeth. Alveolar fractures, along with crown and root fractures and dental luxations, can best be observed with these radiographs (Fig. 2-17). To detect a root fracture, the central x-ray beam must be directed through the fracture line. Multiple radiographs should be taken from several different angles to be more predictable in visualizing fractures.<sup>4,90</sup>

Today, digital radiography is an excellent alternative to conventional radiography, providing an almost instantaneous image, with significantly less radiation, outstanding diagnostic quality, and no processing errors. The storage of the images is only limited by hard drive space. The radiographic image displayed for the patient may allow for better communication regarding diagnosis and treatment plan.<sup>72</sup> Digital radiography is more sensitive than conventional





A



B

**Figure 2-17** **A**, Radiographic evidence of a root fracture of the maxillary right central incisor and **(B)** the maxillary left central incisor.

radiography in the detection of simulated external root resorption lesions.<sup>52,89</sup>

The following should be considered by the clinician during the radiographic examination of a traumatic injury:

- stage of root development
- possible crown and/or root fractures
- relative proximity of the distance between a fractured crown and the pulp



**Figure 2-18** Radiographic evidence of internal root resorption.

- any radiographic abnormalities of the pulp, including pulp calcification or internal resorption (Fig. 2-18)
- possible intraalveolar root fractures
- possible fracture of the alveolar bone
- degree of dislodgment of a luxated tooth from its dental alveolus
- variations in thickness of the periodontal ligament
- signs of root resorption

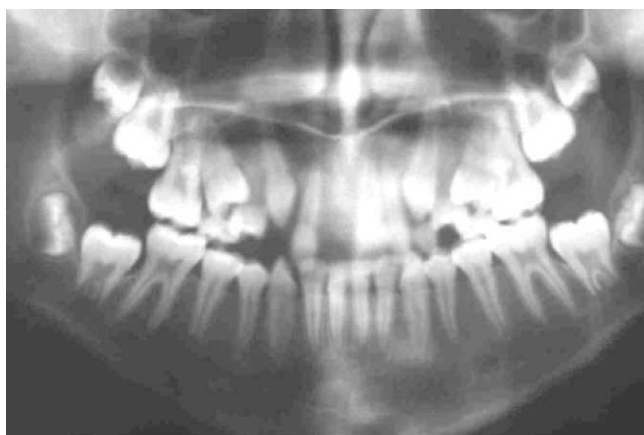
### Alveolar Bone

As described in Chapter 7, radiographs are invaluable when assessing the presence of an alveolar fracture. The panoramic radiograph is an outstanding screening tool for this. It may be necessary to take other radiographs to visualize more details.

## PEDIATRIC CONSIDERATIONS

The clinical approach to a child or adolescent may require different attitudes and considerations than for an adult. Moreover, the clinician should be aware of possible intentional injuries that may have occurred, especially when evaluating traumatic injuries in children.

Although treatment and care are directed to the child, other family members should be included (parents, grandparents, guardian), not just for understanding and permission of the treatment plan, but also for psychological support for the child and for legal reasons (see Chapters 9 and 10). Examination and treatment begin with the clinician using a



**Figure 2-19** Panoramic radiograph reveals avulsion of primary tooth “c” and central incisor.

gentle and considerate approach with the child. This also helps the parents to overcome their fear and anxiety.

The principles of examination are the same for adults and children, but there are additional considerations for children that require specific attention. The main goal for injured primary teeth is to evaluate for possible damage to the permanent tooth buds. Consideration must also be given to the location of the tooth buds when injury involves the facial bones. The pending treatment must assure that no damage to the tooth buds will occur.

Primary incisors loosen more readily following trauma than permanent teeth because of the resilience of the alveolar bone in young children.<sup>6</sup> Furthermore, physiological resorption reduces the crown-root ratio as the tooth approaches the time of natural exfoliation. Only mild trauma may be necessary to loosen these teeth and facilitate an avulsion.<sup>44</sup> During clinical examination of the child with mixed dentition, special attention should be given as to whether the primary tooth was avulsed, intruded, trapped in the mucosal tissues, or previously missing before the traumatic injury (Fig. 2-19).

For young permanent teeth, especially with open apices, preservation of vitality is of utmost importance.<sup>6,21</sup> As discussed, unfortunately the reaction to pulp vitality tests may be misleading and is often of limited value.<sup>32</sup>

With children, the occlusal radiograph may be the only intraoral radiograph possible because of patient compliance. Another radiograph useful for children with compliance issues is an *extraoral* radiograph, using an occlusal film (Fig. 2-20). Cooperation of a parent to support the child may be helpful (Fig. 2-21).

## GERIATRIC CONSIDERATIONS

Special considerations should be given to the medical history when examining an older patient with dental trauma. Aging mostly influences cardiovascular, respiratory, and central



**Figure 2-20** Extraoral radiograph using occlusal film.



**Figure 2-21** Cooperation of a parent to support the child while taking a radiograph.

nervous systems.<sup>68,74,85</sup> Osteoporosis resulting in loss of bone mineral density is common in the elderly, affecting primarily women, which could predispose them to bone fractures.<sup>60</sup>

Older adults typically take more medications.<sup>48,63</sup> For the elderly, there is an “accentuated response of soft tissues to trauma, including ecchymosis, edema, and tissue laxity.”<sup>34</sup> The clinician should also be aware of a potentially missing fixed or removable prosthesis, which may potentially cause airway obstruction.

Fewer nerve branches and reduced vascular supply in aged pulps<sup>13,64</sup> should also be considered when evaluating the vitality of traumatized teeth in elderly patients.

## CONCLUSION

Proper management of the traumatized patient starts with a comprehensive patient assessment and correct diagnosis so that an adequate and timely treatment plan can be implemented. This is important in establishing the most ideal and *safest* dental treatment option. The patient's medical status should be evaluated to ensure that no urgent medical treatment is necessary before managing the dental trauma. A thorough tissue evaluation is imperative, both clinically and radiographically. Proper documentation is necessary with regard to the events that initiated the traumatic injury and for recording all significant findings of the clinical exam. Finally, all patients with traumatic injuries to the head and neck region should be contacted several hours after treatment and again on the following days to check on their status. Patients should be seen if there are any complications, and a referral to their primary care physician is advisable in the event of any adverse change in their medical status.

## REFERENCES

1. al-Qurainy IA, Stassen LF, Dutton GN et al: Diplopia following midfacial fractures, *Br J Oral Maxillofac Surg* 29(5):302-307, 1991.
2. Andersson L, Blomlof L, Lindskog S et al: Tooth ankylosis. Clinical, radiographic and histological assessments, *Int J Oral Surg* 13(5):423-431, 1984.
3. Andreasen FM: Transient apical breakdown and its relation to color and sensibility changes after luxation injuries to teeth, *Endod Dent Traumatol* 2(1):9-19, 1986.
4. Andreasen FM, Andreasen JO: Diagnosis of luxation injuries. The importance of standardized clinical, radiographic and photographic techniques in clinical investigation, *Endod Dent Traumatol* 1:160-169, 1985.
5. Andreasen FM, Pedersen BV: Prognosis of luxated permanent teeth—the development of pulp necrosis, *Endod Dent Traumatol* 1(6):207-220, 1985.
6. Andreasen JO, Andreasen FM, editors: Textbook and color atlas of traumatic injuries to the teeth, ed 3, St Louis, 1994, Mosby.
7. Andreasen JO, Borum MK, Jacobsen HL et al: Replantation of 400 avulsed permanent incisors. 4. Factors related to periodontal ligament healing, *Endod Dent Traumatol* 11(2):76-89, 1995.
8. Andreasen JO, Andreasen FM, Skeie A et al: Effect of treatment delay upon pulp and periodontal healing of traumatic dental injuries—a review article, *Dent Traumatol* 18(3):116-128, 2002.
9. Andresen M, Mackie I, Worthington H: The Periotest in traumatology. Part I. Does it have the properties necessary for use as a clinical device and can the measurements be interpreted? *Dent Traumatol* 19(4):214-217, 2003.
10. Andresen M, Mackie I, Worthington H: The Periotest in traumatology. Part II. The Periotest as a special test for assessing the periodontal status of teeth in children that have suffered trauma, *Dent Traumatol* 19(4):218-220, 2003.
11. Bakland LK, Andreasen JO: Dental traumatology: essential diagnosis and treatment planning, *Endodontic Topics* 7:14-34, 2004.
12. Bender IB, Landau MA, Fonseca S et al: The optimum placement-site of the electrode in electric pulp testing of the 12 anterior teeth, *J Am Dent Assoc* 118(3):305-310, 1989.
13. Bernick S, Nedelman C: Effect of aging on the human pulp, *J Endod* 1(3):88-94, 1975.
14. Bynoe RP, Kerwin AJ, Parker HH III et al: Maxillofacial injuries and life-threatening hemorrhage: treatment with transcatheter arterial embolization, *J Trauma* 55(1):74-79, 2003.
15. Campbell KM, Casas MJ, Kenny DJ et al: Diagnosis of ankylosis in permanent incisors by expert ratings, Periotest and digital sound wave analysis, *Dent Traumatol* 21(4):206-212, 2005.
16. Carranza FA, Takei HH: Clinical diagnosis. In Newman MG, Takei HH, Carranza FA, editors: *Carranza's clinical periodontology*, ed 10, St Louis, 2002, Saunders.
17. Clark DJ, Sheets CG, Paquette JM: Definitive diagnosis of early enamel and dentin cracks based on microscopic evaluation, *J Esthet Restor Dent* 15(7):391-401, 2003.
18. Clark JC, Jones JE: Tooth fragments embedded in soft tissue: a diagnostic consideration, *Quintessence Int* 18(9):653-654, 1987.
19. Cohen S, Liewehr F: Diagnostic procedures. In Cohen S, Burns RC, editors: *Pathways of the pulp*, ed 8, St Louis, 2002, Mosby.
20. Croll TP, Brooks EB, Schut L et al: Rapid neurologic assessment and initial management for the patient with traumatic dental injuries, *J Am Dent Assoc* 100(4):530-534, 1980.
21. Cvek M: Prognosis of luxated nonvital maxillary incisors treated with calcium hydroxide and filled with gutta-percha. A retrospective clinical study, *Endod Dent Traumatol* 8(2):45-55, 1992.
22. Cvek M, Cleaton-Jones PE, Austin JC et al: Pulp reactions to exposure after experimental crown fractures or grinding in adult monkeys, *J Endod* 8(9):391-397, 1982.
23. da Silva AC, de Moraes M, Bastos EG et al: Tooth fragment embedded in the lower lip after dental trauma: case reports, *Dent Traumatol* 21(2):115-120, 2005.
24. Delap TG, Dowling PA, McGilligan T et al: Bilateral pulmonary aspiration of intact teeth following maxillofacial trauma, *Endod Dent Traumatol* 15(4):190-192, 1999.
25. Dewhurst SN, Mason C, Roberts GJ: Emergency treatment of orodental injuries: a review, *Br J Oral Maxillofac Surg* 36(3):165-175, 1998.
26. Dhanrajani PJ, Swaify GA: Aspiration of a bridge and a tooth, *J Craniomaxillofac Surg* 20(2):91-92, 1992.
27. Dorney B: Inappropriate treatment of traumatic dental injuries, *Aust Endod J* 25(2):76-78, 1999.
28. Duus BR, Boesen T, Kruse KV et al: Prognostic signs in the evaluation of patients with minor head injury, *Br J Surg* 80(8):988-991, 1993.
29. Ellis E III, Scott K: Assessment of patients with facial fractures, *Emerg Med Clin North Am* 18(3):411-448, 2000.
30. Feiglin B: Dental pulp response to traumatic injuries—a retrospective analysis with case reports, *Endod Dent Traumatol* 12(1):1-8, 1996.
31. Fulling HJ, Andreasen JO: Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing, *Scand J Dent Res* 84:286-290, 1976.
32. Fuss Z, Trowbridge H, Bender IB et al: Assessment of reliability of electrical and thermal pulp testing agents, *J Endod* 12(7):301-305, 1986.
33. Fuss Z, Tsesis I, Lin S: Root resorption—diagnosis, classification and treatment choices based on stimulation factors, *Dent Traumatol* 19(4):175-182, 2003.
34. Gassner R, Tuli T, Hachl O et al: Cranio-maxillofacial trauma: a 10 year review of 9,543 cases with 21,067 injuries, *J Craniomaxillofac Surg* 31(1):51-61, 2003.
35. Gazelius B, Olgart L, Edwall B: Restored vitality in luxated teeth assessed by laser Doppler flowmeter, *Endod Dent Traumatol* 4(6):265-268, 1988.
36. Gazelius B, Olgart L, Edwall B et al: Non-invasive recording of blood flow in human dental pulp, *Endod Dent Traumatol* 2:219-221, 1986.



37. Goho C: Pulse oximetry evaluation of vitality in primary and immature permanent teeth, *Pediatr Dent* 21(2):125-127, 1999.
38. Gonzalez RP, Fried PO, Bukhalo M et al: Role of clinical examination in screening for blunt cervical spine injury, *J Am Coll Surg* 189(2):152-157, 1999.
39. Gottesman RF, Komotar R, Hillis AE: Neurologic aspects of traumatic brain injury, *Int Rev Psychiatry* 15(4):302-309, 2003.
40. Gunji T: Morphological research on the sensitivity of dentin, *Arch Histol Jpn* 45(1):45-46, 1982.
41. Harran-Ponce E, Holland R, Barreiro-Lois A et al: Consequences of crown fractures with pulpal exposure: histopathological evaluation in dogs, *Dent Traumatol* 18(4):196-205, 2002.
42. Helveston EM: The relationship of extraocular muscle problems to orbital floor fractures: early and late management, *Trans Sect Ophthalmol Am Acad Ophthalmol Otolaryngol* 83(4 Pt 1):660-662, 1977.
43. Herbella FA, Mudo M, Delmonti C et al: "Raccoon eyes" (peri-orbital haematoma) as a sign of skull base fracture, *Injury* 32(10):745-747, 2001.
44. Holan G, Ram D: Aspiration of an avulsed primary incisor: a case report, *Int J Paediatr Dent* 10(2):150-152, 2000.
45. Hyman JJ, Cohen ME: The predictive value of endodontic diagnostic tests, *Oral Surg Oral Med Oral Pathol* 58(3):343-346, 1984.
46. Jackson NG, Waterhouse PJ, Maguire A: Management of dental trauma in primary care: a postal survey of general dental practitioners, *Br Dent J* 198(5):293-297, 2005.
47. Jacobsen I: Criteria for diagnosis of pulp necrosis in traumatized permanent incisors, *Scand J Dent Res* 88(4):306-312, 1980.
48. Jainkittivong A, Aneksuk V, Langlais RP: Medical health and medication use in elderly dental patients, *J Contemp Dent Pract* 5(1):31-41, 2004.
49. Kamboozia AH, Punnia-Moorthy A: The fate of teeth in mandibular fracture lines. A clinical and radiographic follow-up study, *Int J Oral Maxillofac Surg* 22(2):97-101, 1993.
50. Kimberly DR: Unrecognized aspiration of a mandibular incisor, *J Oral Maxillofac Surg* 59(3):350-352, 2001.
51. Kling M, Cvek M, Mejare I: Rate and predictability of pulp revascularization in therapeutically reimplanted permanent incisors, *Endod Dent Traumatol* 2:83-89, 1986.
52. Kositbowornchai S, Sikram S, Nuansakul R et al: Root fracture detection on digital images: eject of the zoom function, *Dent Traumatol* 19:154-159, 2003.
53. Kostopoulou MN, Duggal MS: A study into dentists' knowledge of the treatment of traumatic injuries to young permanent incisors, *Int J Paediatr Dent* 15(1):10-19, 2005.
54. Laks Y, Barzilay Z: Foreign body aspiration in childhood, *Pediatr Emerg Care* 4:102-106, 1988.
55. Lee JY, Yanpiset K, Sigurdsson A et al: Laser Doppler flowmetry for monitoring traumatized teeth, *Dent Traumatol* 17(5):231-235, 2001.
56. Lee ST, Liu TN, Wong YS et al: Relative risk of deterioration after mild closed head injury, *Acta Neurochir Wien* 135:136-140, 1995.
57. Liewehr FR: An inexpensive device for transillumination, *J Endod* 27(2):130-131, 2001.
58. Mackie I, Ghrebi S, Worthington H: Measurement of tooth mobility in children using the Periotest, *Endod Dent Traumatol* 12:120-123, 1996.
59. Mander M, Wencel T, Bazowski P et al: How should we manage children after mild head injury? *Childs Nerv Syst* 16(3):156-160, 2000.
60. Mattson JS, Cerutis DR, Parrish LC: Osteoporosis: a review and its dental implications, *Compend Contin Educ Dent* 23(11):1001-1004, 2002.
61. Mellor TK, Mellor SG: Foreign bodies of dental origin in the appendix, *J R Army Med Corps* 141(3):174-176, 1995.
62. Mesaros SV, Trope M: Revascularization of traumatized teeth assessed by laser Doppler flowmetry: case report, *Endod Dent Traumatol* 13:24-30, 1997.
63. Miller CS, Kaplan AL, Guest GF et al: Documenting medication use in adult dental patients: 1987-1991, *J Am Dent Assoc* 123(11):40-48, 1992.
64. Morse DR: Age-related changes of the dental pulp complex and their relationship to systemic aging, *Oral Surg Oral Med Oral Pathol* 72(6):721-745, 1991.
65. Nee PA, Phillips BM, Bannister CM: Extradural haematoma in a child after an apparently mild head injury, *Br Med J* 306:1665-1666, 1993.
66. Nee PA, Hadfield JM, Yates DW: Significance of vomiting after head injury, *J Neurol Neurosurg Psychiatry* 66(4):470-473, 1999.
67. Oikarinen K, Lahti J, Raustia AM: Prognosis of permanent teeth in the line of mandibular fractures, *Endod Dent Traumatol* 6(4):177-182, 1990.
68. Peacock ME, Carson RE: Frequency of self-reported medical conditions in periodontal patients, *J Periodontol* 66(11):1004-1007, 1995.
69. Petersson K, Soderstrom C, Kiani-Anaraki M et al: Evaluation of the ability of thermal and electrical tests to register pulp vitality, *Endod Dent Traumatol* 15(3):127-131, 1999.
70. Pileggi R, Dumsha TC, Myslinksi NR: The reliability of electric pulp test after concussion injury, *Endod Dent Traumatol* 12(1):16-19, 1996.
71. Pochaczewsky R, Leonidas JC, Feldman F et al: Aspiration and ingested teeth in children, *Clin Radiol* 24:349-353, 1973.
72. Preston JD: Digital radiography—not if, but when, *J Calif Dent Assoc* 27(12):935-941, 1999.
73. Pretto Flores L, De Almeida CS, Casulari LA: Positive predictive values of selected clinical signs associated with skull base fractures, *J Neurosurg Sci* 44(2):77-83, 2000.
74. Ragonesi M, Ivaldi C: Anaesthesiological risk assessment in young/adult and elderly dental patients, *Gerodontology* 22(2):109-111, 2005.
75. Ravin JJ: Follow-up study of permanent incisors with enamel cracks as result of an acute trauma, *Scand J Dent Res* 89(2):117-123, 1981.
76. Rhee P, Nunley MK, Demetriades D et al: Tetanus and trauma: a review and recommendations, *J Trauma* 58(5):1082-1088, 2005.
77. Ritter AM, Muizelaar JP, Barnes T et al: Brain stem blood flow, pupillary response, and outcome in patients with severe head injuries, *Neurosurgery* 44(5):941-948, 1999.
78. Sae-Lim V, Lim LP: Dental trauma management awareness of Singapore pre-school teachers, *Dent Traumatol* 17(2):71-76, 2001.
79. Sae-Lim V, Chulaluk K, Lim LP: Patient and parental awareness of the importance of immediate management of traumatized teeth, *Endod Dent Traumatol* 15(1):37-41, 1999.
80. Sakamoto T, Takasu A, Saitoh D et al: Ionized magnesium in the cerebrospinal fluid of patients with head injuries, *J Trauma* 58(6):1103-1109, 2005.
81. Seltzer S, Bender I, Ziontz M: The dynamic of pulp inflammation: correlations between diagnostic data and actual histologic findings in the pulp, *Oral Surg Oral Med Oral Pathol* 16:846, 1963.
82. Steelman R, Millman E, Steiner M: Aspiration of a primary tooth in a patient with a tracheostomy, *Spec Care Dent* 17:97-99, 1997.
83. Stone CK, Humphries RL: *Current emergency diagnosis and treatment*, ed 5, New York, 2003, McGraw-Hill Medical.
84. Teasdale G, Murray G, Parker L et al: Adding up the Glasgow Coma Score, *Acta Neurochir Suppl (Wien)* 28(1):13-16, 1979.

85. Umino M, Nagao M: Systemic diseases in elderly dental patients, *Int Dent J* 43(3):213-218, 1993.
86. Vincent JL, Berre J: Primer on medical management of severe brain injury, *Crit Care Med* 33(6):1392-1399, 2005.
87. Vongsavan N, Matthews B: Experiments on extracted teeth into the validity of using laser Doppler techniques for recording pulpal blood flow, *Arch Oral Biol* 38(5):431-439, 1993.
88. Welbury RR, Murphy JM: The dental practitioner's role in protecting children from abuse, 2. The orofacial signs of abuse, *Br Dent J* 24;184(2):61-65, 1998.
89. Westphalen VP, Gomes de Moraes I, Westphalen FH et al: Conventional and digital radiographic methods in the detection of simulated external root resorptions: a comparative study, *Dentomaxillofac Radiol* 33(4):233-235, 2004.
90. Whaites E: Essentials of dental radiography and radiology, ed 3, London, 2002, Churchill Livingstone.
91. Yee KF, Cravens J, Kotler R: Traumatic ophthalmoplegia: a complication of fractures of the zygomaticomaxillary complex. Review of etiology with report of an unusual case, *Laryngoscope* 85(3):570-575, 1975.

# CROWN FRACTURES: A PRACTICAL APPROACH FOR THE CLINICIAN



ANNA B. FUKS AND JOE CAMP

## CHAPTER OUTLINE

### CLASSIFICATION

### ETIOLOGICAL FACTORS

### DIAGNOSIS AND CLINICAL FINDINGS

### RADIOGRAPHIC FINDINGS

### BIOLOGICAL CONSIDERATIONS AND TREATMENT PRINCIPLES

### ENAMEL INFRACTIONS AND ENAMEL FRACTURES

#### Diagnosis

#### Histopathology

#### Treatment

#### Prognosis

### ENAMEL-DENTIN FRACTURE WITHOUT PULP

#### INVOLVEMENT (UNCOMPLICATED CROWN FRACTURES)

#### Diagnosis

#### Histopathology

#### Treatment

##### Restoration (Composite Buildup)

##### Reattachment of Coronal Segment

#### Prognosis

##### Restoration by Composite Buildup

##### Reattachment of Coronal Segment

### ENAMEL-DENTIN FRACTURE WITH PULP INVOLVEMENT (COMPLICATED CROWN FRACTURES)

#### Diagnosis

#### Histopathology

#### Factors Influencing Choice of Treatment

#### Treatment: Mature and Immature Teeth

##### Direct Pulp Capping

##### Technique

#### Pulpotomy (Partial or Coronal)

#### Coronal Pulpotomy

#### Deep Pulpotomy

#### Follow-up to Pulp Capping and Pulpotomy

#### Pulp-Capping Agents

#### Prognosis

### TREATMENT TECHNIQUES FOR NONVITAL TEETH

#### Mature Teeth

#### Immature Teeth

##### Apexification

##### Apexification: Technique

##### Apical Barrier: Technique

### RESTORATION OF IMMATURE ROOTS

### CROWN FRACTURES IN PRIMARY TEETH

#### Clinical Appearance

#### Radiographic Appearance

#### Biological Considerations

#### Uncomplicated Crown Fractures (No Pulp Exposure)

#### Complicated Crown Fractures (Pulp Exposure)

##### Incidence

##### Treatment Options

##### Direct Pulp Capping: Technique

##### Partial Pulpotomy or Cvek Technique

##### Pulpotomy (Partial/Coronal)

##### Pulpectomy

##### Extraction



Crown fractures account for the highest percentage of all traumatic injuries to the permanent dentition (between 26% and 76% of the dental injuries that have been reported in the dental literature).<sup>10,11,41,136,139</sup> Root fractures occur at a much smaller rate, between 0.3% and 5%.<sup>11</sup>

The prognosis for a traumatized tooth depends mainly on the accuracy of the diagnosis and on the choice of the appropriate treatment at two specific levels: at the pulpal level and at the restorative level.<sup>93</sup> On the pulpal level, the clinician has to have a good knowledge of how the pulp reacts to trauma and what effects are induced by the clinical procedures. On the restorative level, the clinician must be aware of the new developments in adhesives and restorative materials while being conscious of some technical limitations.

The aim of this chapter is to report the recent knowledge based on experimental, epidemiological, histopathological, and clinical studies regarding pulp reactions to crown fractures, and to present up to date clinical restorative techniques.

## CLASSIFICATION

The World Health Organization (WHO) in 1995 published the *Application of the International Classification of Diseases to Dentistry and Stomatology*, with a comprehensive classification of fractures of the teeth.<sup>138</sup>

- a. Enamel infraction and enamel fracture only (S.02.50)
- b. Fracture of enamel and dentin (uncomplicated crown fractures, S.02.51)
- c. Fracture of crown of the tooth with pulp involvement (complicated crown fracture, S.02.52)

## ETIOLOGICAL FACTORS

The most common causal factors of crown or crown-root fractures in the permanent dentition are injuries caused by falls, contact sports, car crashes, or foreign bodies hitting the teeth.<sup>10</sup> Occupational hazards may also play a role. For example, enamel fractures are very common among glass blowers, probably because of the impact of the blowpipe.<sup>110</sup>

## DIAGNOSIS AND CLINICAL FINDINGS

*Enamel infractions* are very common but frequently overlooked.<sup>29</sup> They appear as cracks within the enamel that do not cross the dentinoenamel junction. These injuries are caused by direct impact to the enamel, as in falls or vehicular accidents, explaining their frequent appearance on the labial surface of the maxillary incisors.<sup>102,135</sup> Infractions are frequently the only evidence of trauma, but they can also be associated with other injuries. Thus *the presence of infraction*

*lines or cracks on the tooth should draw the clinician's attention to the possible presence of other injuries, particularly to the supporting structures.*<sup>102</sup>

*Fractures without pulpal involvement* occur more frequently than those with a pulp exposure, both in primary and permanent teeth,<sup>7,55</sup> and affect mainly the maxillary incisors.<sup>16</sup> Similar to enamel infractions, *crown fractures* can be associated with luxation injuries, affecting the prognosis.<sup>130</sup> Clinical examination of the fractured teeth should be done after a thorough cleaning to assess the extent of the fracture and detect the possibility of minute pulp exposures.

Crown fractures are usually sensitive to thermal changes; sometimes the layer of dentin covering the pulp is so thin that one can discern its pinkish hue. In these cases, care should be taken not to expose the pulp with the probe while checking for exposures.<sup>10</sup>

*Crown fractures with pulp exposures* (complicated crown fractures) frequently present a slight hemorrhage from the exposure site. A pulp polyp can be produced when treatment is delayed.<sup>13,14</sup>

## RADIOGRAPHIC FINDINGS

The radiographic examination completes the clinical evaluation and provides information, including the size of the pulp, stage of root development, presence of a concomitant root fracture, or luxation. All of these parameters can influence the future treatment.<sup>8,9</sup>

The preoperative diagnostic radiographs serve as a base for comparison with follow up radiographs and are particularly useful to disclose the presence of a hard tissue barrier over a treated exposed pulp. Clinically the pulp cavity is usually larger and the pulp horns are closer to the incisal edge than it appears radiographically.<sup>10</sup>

## BIOLOGICAL CONSIDERATIONS AND TREATMENT PRINCIPLES

Although there are no histological studies on the state of the pulp after enamel infractions or enamel fractures occur, some researchers<sup>93</sup> assume that there might be a transient inflammatory phase, a transient depolarization of nerve fibers in the apical region (negative response to pulp sensitivity testing), and/or a local hemorrhage (change in color).<sup>97</sup>

When the fracture involves enamel and dentin, a large number of dentinal tubules are exposed. It has been estimated that an exposure of 1 square millimeter of dentin would expose 20,000 to 65,000 dentinal tubules.<sup>84</sup>

The exposed dentinal tubules are an easy pathway for bacteria and their toxins to invade the pulp and result in pulpal inflammation.<sup>10</sup> Investigators<sup>80</sup> left prepared dentin open to

saliva and plaque accumulation in vivo, and observed that the rate of bacterial penetration was 0.52 mm after approximately 84 days. It has also been demonstrated that bacterial penetration is faster in dentin exposed by trauma than in the dentin exposed by grinding, probably because of the absence of a smear layer in the fractured dentin.<sup>92,131</sup> Another factor that can increase the speed of bacterial penetration is the lack of hydrostatic pressure from an outward pulpal fluid flow that is minimal or nonexistent, as in cases of a compromised blood supply.<sup>82,92,132–134</sup>

The severity of the pulpal response is related to pulpal vascularity—mainly whether or not the neurovascular supply has been compromised by a concomitant luxation injury.<sup>6</sup> Treatment should consist principally of protecting the pulp from external injuries, and restoring normal function and esthetics. If no concomitant luxation injury is present, the tooth can be restored immediately, using conventional composite restorative techniques. When the concomitant luxation injury is associated with tooth mobility and bleeding from the sulcus, a temporary restoration is recommended because it is difficult to keep a dry field. In these cases, care should be taken not to push the temporary material into the ruptured periodontal ligament space.<sup>13</sup>

## ENAMEL INFRACTIONS AND ENAMEL FRACTURES

### DIAGNOSIS

Infractions are often missed with reflected illumination, but are easily disclosed by directing the light beam perpendicular to the long axis of the tooth from the incisal edge (transillumination).<sup>13,18</sup> Fiber optics are also useful to detect these injuries. Differential diagnosis should exclude cracks caused by wide, rapid thermal changes (from hot coffee to cold ice cream).<sup>28</sup>

Enamel fractures can be diagnosed by direct clinical examination of the missing tooth structure from the crown. They occur mainly in the anterior region, at the incisal edge or at a proximal angle.<sup>93</sup> The tooth is usually not sensitive to variations in temperature, dehydration, or pressure. However, pulp testing can be temporarily negative or there might be a change in color, particularly in cases with concomitant luxation injuries.<sup>13</sup>

### HISTOPATHOLOGY

Enamel infractions can be seen in ground sections, and they appear as dark lines running parallel to the enamel prisms, terminating at the dentinoenamel junction.

As reported above, there are no studies dealing with the histology of the pulp after enamel infractions or enamel fractures.<sup>93</sup>

### TREATMENT

Enamel infractions as a rule do not require treatment. However, in severe cases of multiple infraction lines, the indication might be to seal the enamel surface with an adhesive to prevent taking up stains from tobacco, food, drinks (tea, red wine, cola), or other liquids like chlorhexidine mouthwashes.<sup>13</sup> One investigator<sup>81</sup> found in an in vitro model that enamel-dentin cracks might be a portal of entry for bacterial invasion in apparently intact teeth.<sup>102</sup>

Sealing of cracked teeth may be performed with any adhesive system after appropriate cleaning and acid etching. Alternatively, to simplify the clinical procedures and save time, the new self-conditioning systems like Adper L-Pop (3M Espe, USA) can be used.<sup>93</sup>

Treatment of crown fractures confined to the enamel will depend on the amount of tissue lost. Simple contouring might be enough; in more extensive crown fractures involving the proximal-incisal edge, particularly for midline symmetry, a fractured mesial corner will preclude recontouring, and a restoration becomes necessary.<sup>10</sup>

### PROGNOSIS

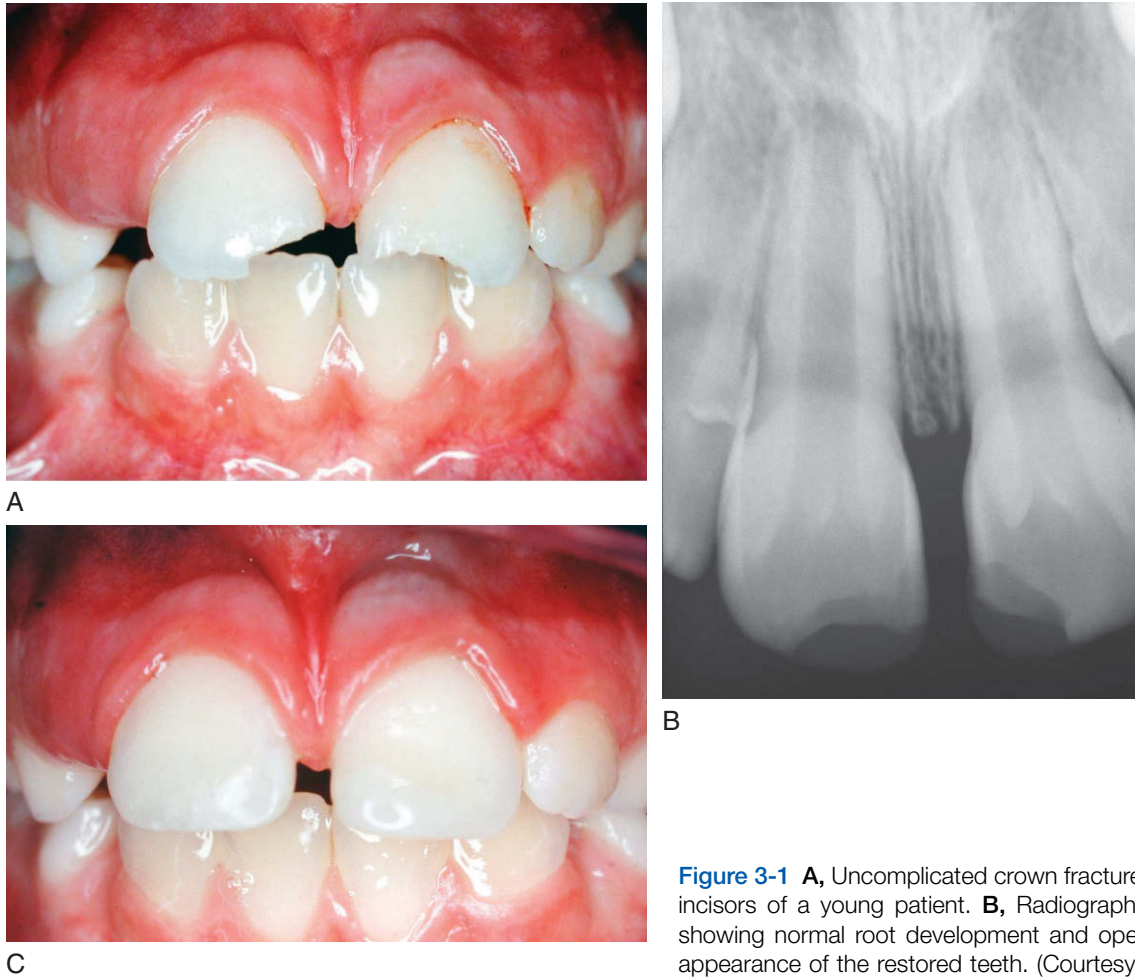
The prognosis is very good for both types of injuries.<sup>25,102,103,120</sup> The likelihood of pulp survival after enamel infractions ranges from 97% to 100%, and after enamel fractures it ranges from 99% to 100%.<sup>93</sup> These few cases of pulp necrosis may be due to concomitant concussion or subluxation injuries that might have been overlooked.<sup>93</sup>

## ENAMEL-DENTIN FRACTURE WITHOUT PULP INVOLVEMENT (UNCOMPLICATED CROWN FRACTURES)

### DIAGNOSIS

The fractured tooth should be cleaned with water spray or a wet cotton roll before examination. The extent of the fracture is then assessed by checking the missing tooth structure, looking carefully for the presence of minute pulp exposures. The tooth is generally sensitive to variations in temperature, dehydration, and pressure because of the exposure of sectioned dentinal tubules. This sensitivity is greater in younger teeth and in fractures closer to the pulp because the number and diameter of the tubules are higher in younger patients.<sup>96</sup>

Similar to what was described for enamel fractures, pulp testing can be temporarily negative, or there might be a change in color, particularly in cases with concomitant luxation injuries.<sup>13</sup>



**Figure 3-1** **A**, Uncomplicated crown fractures of maxillary central incisors of a young patient. **B**, Radiograph of the injured teeth showing normal root development and open apices. **C**, Clinical appearance of the restored teeth. (Courtesy Dr. Lucia Blanco.)

## HISTOPATHOLOGY

The clinician must be aware of the histophysiological and histopathological basics to understand the mechanisms of reaction of the tooth to trauma and other injuries.

Bacteria and bacterial byproducts have been proven to be the most important etiological factors in inflammatory reactions of the human dental pulp. Moreover, it has been demonstrated that clinical pulp inflammation cannot develop without the presence of bacterial infection.<sup>19,32</sup> In crown fractures, bacteria can invade the sectioned dentinal tubules, producing inflammation.

The pulp has the potential to defend itself by means of two defensive mechanisms:

1. *Passive mechanism*—consisting of an outward flow of dentinal fluid within the tubules that resists the invasion of bacteria through a gradient of hydrostatic pressure.<sup>28</sup>
2. *Active mechanism*—consisting of the ability of the pulp to react immediately with an inflammatory response to outside stimuli, to bacterial toxins, or to bacteria through the blood circulation. Any factor that can affect these protective mechanisms, such as impairment of pulpal circulation (as in cases of concomitant luxation) or the age of

the tooth (as in its potential for repair), will have to be taken into consideration when doing the emergency treatment and for assessing the prognosis of the traumatized tooth.<sup>93</sup>

## TREATMENT

### Restoration (Composite Buildup)

One of the major challenges a dentist has to face in clinical practice is to restore the esthetics and function of a traumatized tooth and to maintain the vitality of the pulp. While restoring the proper shape and dimension of the tooth, several factors have to be taken into consideration. These include the shade, opacity, translucency, and more recently the appropriate fluorescence and opalescence.<sup>93</sup>

The composite resins available today, in conjunction with a careful technique, allow for achievement of excellent esthetic and functional results (Fig. 3-1). Presently the technique that offers the most predictable results recommends the use of a silicone reference guide. Using this guide, the clinician can assess the thickness and the size of the different increments of composite to be applied, employing a more



opaque composite in the dentin area and a more translucent shade for the incisal edge. The final shape and texture can be achieved with appropriate composite finishing procedures. The step by step technique is presented in Figure 3-2.

### Reattachment of Coronal Segment

An alternative treatment option for restoring fractured incisors is bonding of the chipped fragment onto the crown. The first reference to the use of this technique appeared in 1964, when clinicians described a case of rehabilitation of a fractured incisor using the patient's natural crown.<sup>26</sup> However, only a few years later another clinician<sup>115,116</sup> published a protocol for fragment and tooth preparation, using an acid etch and composite technique. With the new dentin bonding agents available today, fragment reattachment is becoming more and more attractive, mainly as a result of several advantages: psychological acceptance by the patient and/or parents, reduced chair time, accurate restoration of tooth morphology and texture, and using the natural tooth, the abrasion would be similar to that of the contralateral and opposing teeth.<sup>93</sup> This technique can be preferred over a composite buildup when the fragment is available and of reasonable size, and its adaptation to the tooth is accurate. Bigger fragments are easier to bond; small or multiple fragments are difficult to manage.

Other investigators<sup>93</sup> proposed a technique to improve fragment retention: the use of a groove in the fragment, which would increase the bonding surface, and thus the bonding strength. These authors recommend making a small groove with a round diamond bur within the dentin at the fractured interface. The enamel is then acid etched for 30 seconds and the dentin for 10 seconds with a 35% phosphoric acid gel. After conditioning the dentin, the prepared fragment is left uncured, in the absence of light; the fractured tooth is prepared the same way. A resin composite is then applied into the groove of the fragment and fitted against the fractured surface. Excess is removed before curing the composite for 40 seconds, both buccal and lingual. Polishing is performed in a conventional manner using diamond burs, discs, and polishing strips where necessary (Fig. 3-3).

## PROGNOSIS

### Restoration by Composite Buildup

The long term prognosis of class IV (involving the incisal angle) composite restorations still remains questionable, with different results concerning durability and esthetics reported in various studies.<sup>23,42,106,117,118</sup> In an interesting article assessing class IV restorations, investigators<sup>104</sup> reported that over a period of 15 years, 19% of the restorations were changed 10 times and that 25% were rated unsatisfactory at the final examination.

However, other investigators<sup>23</sup> reported that the main reason for failures were related to the adhesive system used (bond failure leading to composite fracture). This would

cause marginal microleakage, which not only produces marginal discoloration, but also causes the penetration of microorganisms into the dentinal tubules with the subsequent contamination of the dental pulp.<sup>88,89,109</sup> Other reasons for restoration failures reported by these authors are cohesive fractures of the composite, shade instability, recurrent decay, or new trauma suffered by the patient. According to these authors,<sup>23</sup> the mean time for restoration replacement is about 5 years. In the last decade, with the introduction of latest generation adhesive systems and improvement of the properties of the composites, an increase in the longevity of these restorations has been observed.<sup>42</sup>

### Reattachment of Coronal Segment

A long-term extensive multicenter clinical study<sup>12</sup> demonstrated that fragment retention was much higher when using a total etch technique and dentin bonding than when using enamel etching alone (50% retention for the total etch at 30 months, and for the enamel etch at 12 months). These authors also pointed out that reinforcement of the fracture line did not prolong fragment retention.<sup>12</sup> The main causes for fragment loss were new trauma, nonphysiological use of the restoration, and horizontal traction. One of the most important findings of this study was that the results from fragment bonding versus composite buildup did not differ significantly. These results seem somewhat low for the composite group, as success rates of composite buildup have been shown to be much higher.<sup>42</sup>

Preliminary results of a scanning electrical microscope (SEM) study reveal that no gap was detected at the dentin-composite interface with the use of a groove, whereas some gap was seen when no groove was performed.<sup>93</sup>

Fragment bonding apparently has no deleterious effect on the pulp. In experimentally induced crown fractures in monkeys, researchers<sup>105</sup> reported that the pulps were in good condition, and hard tissue formation was observed after 3 months. These findings reinforce those reported in the clinical study of other investigators,<sup>12</sup> which showed the absence of pulp necrosis as long as there was no concomitant luxation injury.

## ENAMEL-DENTIN FRACTURE WITH PULP INVOLVEMENT (COMPLICATED CROWN FRACTURES)

### DIAGNOSIS

Diagnosis is made by clinical examination of the missing tooth structure of the crown and the presence of a pulp exposure. Usually, slight hemorrhage from the exposed part of the pulp is present. Proliferation of pulp tissue (pulp polyp) can occur when treatment in young teeth is delayed.<sup>10</sup>



A



B



C



D



E



F

**Figure 3-2** Restoration of a fractured incisor by composite buildup technique. **A**, Take alginate or silicone impression and pour plaster model. Send to lab technician to fabricate mouthguard. **B**, Select appropriate shades for dentin and enamel. **C**, Place mouthguard in position and section in mesial to allow placement of matrix. **D**, Protect adjacent tooth with adhesive strip and acid etch the tooth to be restored. **E**, Place matrix band on mouthguard to establish mesial contour. **F**, Build up restoration starting palatal, adding dentin colors followed by enamel shades until completing the restoration.





G

**Figure 3-2, cont'd G,** Finish and polish with conventional techniques. (Courtesy Dr. Israel Varsano.)

Depending on the *absence* or *presence* of a concomitant luxation injury, the pulp will have a bright red, cyanotic, or ischemic appearance, respectively.<sup>13</sup> The tooth is generally sensitive to variations of temperature, dehydration, and pressure caused by the sectioning of the dentinal tubules and of the exposed pulp. The response to pulp testing is usually positive, unless there is a concomitant luxation injury.

The radiographic examination adds important information to the clinical evaluation (such as the stage of root development or the size of the pulp) and helps in determining the type of treatment to be undertaken. Moreover, the radiograph serves as a record for evaluating the success of the treatment, particularly for evaluating root maturation and the presence of a hard tissue barrier covering the exposed area.<sup>22</sup>



A



B



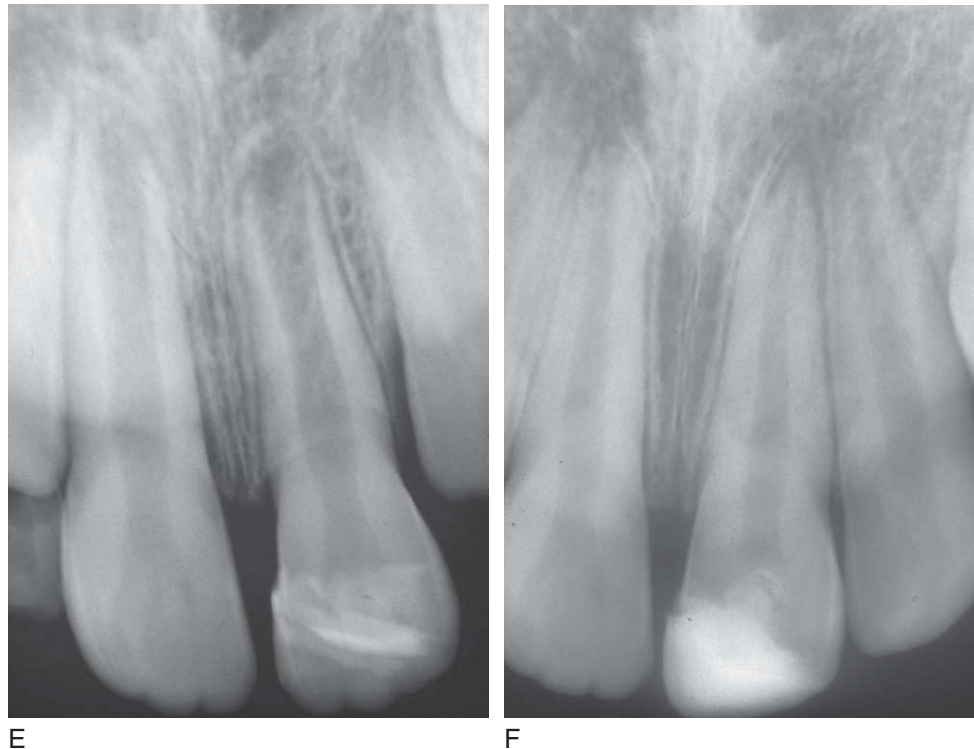
C



D

**Figure 3-3 A,** Uncomplicated crown fracture in the upper left central incisor of a 9-year-old boy. **B,** View of the coronal dental fragment. **C,** Preoperative radiograph showing the line of fracture. Note the apex more open than the other central incisor. **D,** The dental fragment was joined with a bonding technique.





**Figure 3-3, cont'd** **E**, Postoperative radiograph: the patient had a second fall 1 year later and Cvek's technique was performed. **F**, Radiograph taken 4 years after the first trauma and 2 years after partial pulpotomy; both teeth are vital and the apex of the left central incisor is closed. (Courtesy Dr. Lucia Blanco.)

## HISTOPATHOLOGY

Exposed dentinal tubules and the exposed pulp cause indirect and direct injury to the pulp, which reacts with an inflammatory response, followed by the subsequent formation of granulation tissue.<sup>40</sup> Bacteria can penetrate the exposed dentinal tubules and can also be found on the surface of the pulp. However, bacterial colonization is impeded if pulpal vascularity is intact.<sup>13</sup>

## FACTORS INFLUENCING CHOICE OF TREATMENT

It is now well established that teeth have the potential to form a hard tissue barrier subsequent to a pulp exposure when a biocompatible pulp-capping agent is placed over it. The aim of treatment should be the preservation of a vital, noninflamed pulp, biologically walled off by a continuous tissue barrier. In most cases, this can be achieved by pulp capping, partial pulpotomy, coronal pulpotomy, or deep pulpotomy.<sup>40</sup>

The stage of root development, the size of the exposure, and the time elapsed between the injury and the emergency treatment seem to be the most important factors to determine the type of treatment to be undertaken. Several other factors, such as the health of the pulp before the trauma, the

age of the patient, the presence of a concomitant luxation injury, the effect of the surgical procedures, and the type of pulp-capping agent employed may also influence the selection of the most appropriate treatment.<sup>40</sup>

- *Stage of root development* is by far the most important factor to be considered. It is generally accepted that the pulp should be maintained in cases of immature teeth with open apices, whereas it can be removed in teeth with closed apices. However, Cvek<sup>40</sup> recommends maintaining the vitality of teeth in children and adolescents, even those with closed apices, because pulp removal deprives the tooth of physiological dentin apposition, leaving the tooth with thin dentinal walls and more prone to future fractures.
- *Size of the exposure*: Investigators<sup>93</sup> claim that the larger the exposure, the lower the chance of healing through the formation of a dentin bridge. These authors believe that 1.5 mm diameter of pulp opening is the higher limit for a reasonable success rate, although there is no literature confirming this. Cvek, however, showed that the size of the exposure and the time elapsed since the injury will determine the level of pulp amputation, and concluded that these factors are not critical for healing if only superficial layers of the pulp are removed.<sup>22,40,46</sup>
- *Presence or absence of concomitant luxation injuries*: A concomitant luxation injury compromises the nutritional

supply to the pulp and may lead to pulp necrosis, contraindicating, in principle, conservative pulp-preserving treatments. However, in immature luxated teeth, the chance of pulp survival is considerable, and conservative treatment may allow further root development to occur. Thus treatment should be determined according to the severity of the injury to the periodontal ligament and to the maturity of the tooth. Two studies by the same investigators<sup>10,14</sup> showed that in teeth with enamel-dentin fractures that do not involve the pulp, the incidence of pulp necrosis increases from 5% to up to 75% for teeth with closed apices, when a concomitant luxation injury is present.

- *Pulpal health before trauma:* In teeth that had previous trauma or large carious lesions, the potential for healing is diminished,<sup>17</sup> and care should be taken when recommending conservative treatment for those teeth.
- *Age of the tooth:* The effect of age is controversial. A diminished response to injury has been observed in pulps of old rats when compared with young ones.<sup>114</sup> However, successful treatment following pulp exposure caused by caries or trauma in older patients has been reported. Conversely, it is known that for various reasons, degenerative change in the pulp increases with age.<sup>20,40</sup> Thus although there is no age limit for conservative treatments, pulp capping or pulpotomy (both partial and coronal) should *not* be performed if degenerative or inflammatory changes are anticipated, such as teeth with calcific metamorphosis of the pulp or teeth that are periodontally involved in adults.<sup>121</sup>

## TREATMENT: MATURE AND IMMATURE TEETH

Pulp capping and pulpotomy procedures should be attempted whenever possible in teeth having immature apices with crown fractures that expose the pulp. The use of these conservative treatments does not preclude the use of more extensive procedures in the event of nonhealing.

Many materials and drugs have been used for pulp-capping agents. Traditionally the application of calcium hydroxide to stimulate dentin bridge formation has been the treatment of choice. However, better results have been reported<sup>1,66,98</sup> using MTA (mineral trioxide aggregate) (Pro Root MTA™, Dentsply Tulsa Dental, Tulsa, Okla.). The various pulp-capping agents and their effect on pulp healing will be described further in this chapter.

### Direct Pulp Capping

Pulp capping should be confined to small exposures and performed within the first 24 hours after injury. A restoration over the pulp cap must be placed to ensure a tight seal against bacterial invasion.<sup>10,14</sup> It is thought that in small exposures treated soon after the injury, the mechanical damage and inflammation in the pulp cannot be deeper than

the necrotizing effect of calcium hydroxide or MTA over healthy pulp tissue because the bacteria on the dentin fracture and on the wound surface will otherwise not be eliminated by the action of the pulp capping drug.<sup>40</sup>

**Technique** The fracture surface and the pulpal wound should be cleaned with saline or diluted in 2.5% sodium hypochlorite<sup>49</sup> to disinfect the cavity and remove any blood clot that might be present. When bleeding has stopped, the exposed pulp is covered with the selected pulp dressing (soft or hard setting calcium hydroxide, MTA). If bleeding persists, application of light pressure to the exposure site with a cotton pellet moistened with saline will stop it. If bleeding continues, direct pulp capping should not be performed and a coronal pulpotomy should be considered.

The pulp capping agent is then covered with a glass ionomer cement (in the case of a soft setting calcium hydroxide or MTA), and the tooth is restored with a leak-free composite restoration.

### Pulpotomy (Partial or Coronal)

With longer periods of time subsequent to the trauma, or with larger exposures, a partial pulpotomy (Cvek technique) or coronal pulpotomy should be performed. Pulpotomy involves the removal of damaged and inflamed tissue to the level of a clinically healthy pulp, followed by a pulp dressing (calcium hydroxide or MTA). Depending on the size of the exposure and the time elapsed since the injury, different levels of pulpal amputation have been recommended (partial or coronal pulpotomy<sup>40</sup>). As mentioned previously, neither the size of the exposure nor the time interval between the trauma and the treatment is critical for healing when only superficial layers of the pulp are amputated.<sup>40,46</sup> Thus when the pulp is vital and hyperplastic tissue is seen on the exposure site, only superficial layers of the pulp and surrounding dentin should be removed (partial pulpotomy). This treatment is suitable for both immature and mature teeth.

The tooth should be anesthetized and isolated with a rubber dam. If any loosening of the tooth has occurred, the rubber dam clamp must be applied to adjacent uninjured teeth. If injury to the tooth or incomplete eruption precludes the placement of the rubber dam, then isolation with cotton rolls, protection of the throat with gauze, and constant aspiration by a dental assistant may be used to maintain a protected and dry field. Pulpal amputation and placement of the capping material must be achieved in an aseptic environment to prevent further contamination of the pulp during the procedure. Cvek<sup>36</sup> has shown that pulp exposures from traumatic injuries usually exhibit a proliferative reaction, with inflammation extending only a few millimeters into the tissue. Further data obtained from animal studies<sup>37,59</sup> in which the pulps were intentionally exposed by fracture or grinding have confirmed that pulpal changes are characterized by a proliferative response with inflammation extending no more than 2 mm from the exposure site.

In the Cvek pulpotomy, only tissue judged to be inflamed is removed—usually approximately 1 to 2 mm below the

level of the pulp exposure. Cutting of the tissue with an abrasive high speed diamond bur with water cooling has been shown to be the least damaging to the underlying tissue.<sup>54</sup> Replacing the diamond by a carbide #330 bur appears to have similar results.<sup>21</sup> After pulp amputation, the preparation is thoroughly washed with saline and dried with cotton pellets. Spoon excavators are contraindicated because these tend to tear out larger segments of tissue. The preparation is washed with physiological saline or sterile water to remove debris.

Hemorrhage is controlled by placing sterile cotton pellets, which are moistened with sterile saline, on the amputation site and applying slight pressure with additional dry pellets. Hemorrhage should be controlled after a few minutes. If hemorrhage continues, the amputation site is checked to ensure the removal of all debris and pulpal tissue remnants coronal to the pulp stump. Air should not be blown on the exposed pulp because it may cause tissue damage by desiccation.

Diluted sodium hypochlorite has been reported as a hemostatic agent in pulpotomy studies.<sup>5,34</sup> The solution was not toxic to pulp cells, or inhibitory to healing, odontoblastoid cell formation, or dentinal bridge formation. Cotton pellets are moistened in 2.5% sodium hypochlorite and placed against the pulp stumps for 20 to 50 seconds. If hemorrhage persists, amputation is performed at a more apical level. Bleeding can be also controlled by flushing continuously with sterile solution to prevent clot formation.<sup>22</sup> Once hemorrhage is controlled, a dressing of calcium hydroxide is gently placed over the amputation site or, alternatively, the pulp can be covered with a layer of MTA (Fig. 3-4). If calcium hydroxide is used, care should be taken not to push the material into the pulp because it might cause inflammation, increasing the potential for nonhealing of the procedure. However, if the pulpotomy is successful, this material may create a tendency for calcific obliteration of the pulp. A creamy mix of zinc oxide and eugenol or other cement is flowed over the calcium hydroxide and allowed to set. The teeth must be examined clinically and radiographically every 3 months to assess healing.

The tooth should remain asymptomatic and retain normal pulp sensitivity. Radiographically the formation of a hard tissue barrier should be observed, and root development, especially the immature root, should progress normally.

After 3 months, Cvek proposes that the hard tissue dentinal barrier must be clinically monitored and the final restoration placed in a timely manner<sup>40</sup> (see Fig. 3-3).

When a composite resin is planned for restoration of the tooth, eugenol-containing compounds must be avoided because they can interfere with the setting reactions of the composite. In these cases, a layer of glass ionomer is placed over the zinc oxide-eugenol.

If MTA is the selected pulp-capping agent, it is mixed and picked up in a small amalgam carrier. The MTA pellet is partially extruded from the carrier, retaining 1 to 1½ mm inside.

The remaining MTA is gently placed over the pulp stump and teased into place with a cotton pellet in endodontic pliers. The layer of MTA should be approximately 1 mm in depth. A thin layer of “flowable” glass ionomer or composite resin is placed over the MTA and light cured. The tooth can then be restored with an acid etched composite restoration without disturbing the MTA (see Fig. 3-4).

In the last few years, MTA has been successfully employed in pulpotomies in several human and animal studies.<sup>43,61,63</sup>

### Coronal Pulpotomy

In immature permanent teeth, when the inflammation has progressed deeper than 2 mm from the exposure site, a coronal pulpotomy is performed to allow completion of root development; it may be regarded as an apexogenesis procedure. Pulpotomy is performed in teeth in which it is assumed that healthy pulp tissue, with a potential to produce a dentin bridge and complete the formation of the root, remains in the root canal. The technique for coronal pulpotomy in permanent teeth is similar to that for primary teeth, and the dressing material should maintain pulp vitality and function. Care should be taken to remove the blood clot before placement of a calcium hydroxide paste over the pulp stumps because its presence may compromise the treatment outcome. It has been demonstrated that leaving the blood clot may result in the formation of dystrophic calcifications and internal resorption. The blood clot may also interfere with dentin bridge formation and serve as a substrate for bacteria if a leaky restoration is present.<sup>112</sup>

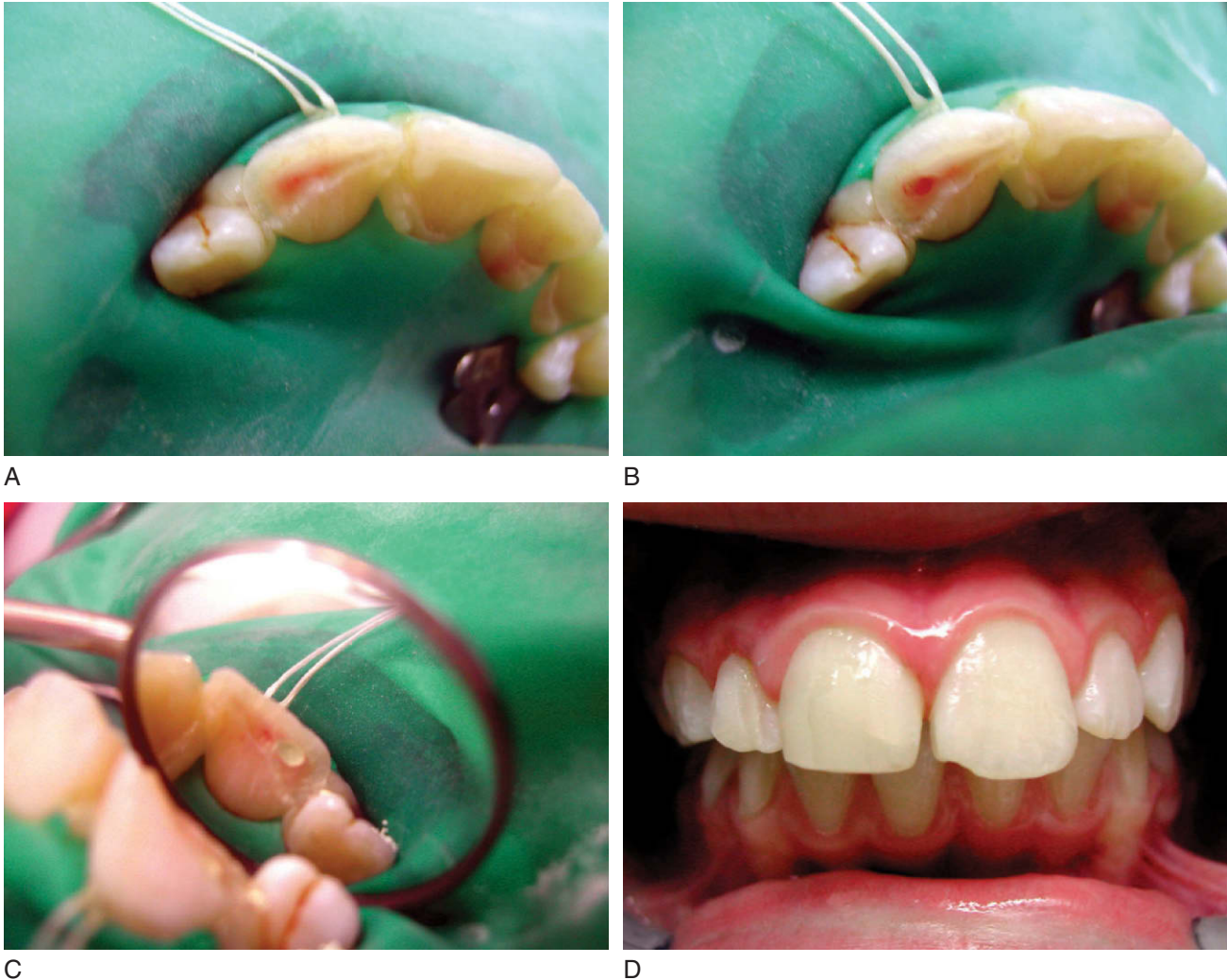
When proper healing occurs, a dentin bridge may form under the calcium hydroxide dressing, indicating that the pulp tissue is normal with a dentinogenic potential, allowing further root development. Sometimes the calcium hydroxide dressing may form dystrophic calcifications, rendering the root canal difficult to negotiate, in the event that endodontic treatment becomes necessary.<sup>10</sup> Clinical and radiographic follow-up of these teeth is essential to ensure that pulpal or periapical pathosis is not developing. Providing a timely permanent restoration is of utmost importance (Fig. 3-5).

Recently there have been successful reports of pulpotomies using MTA in primary teeth.<sup>2,4,43,61</sup>

### Deep Pulpotomy

This treatment should be considered in teeth with immature apices with pulp exposures in which treatment was delayed, and it became necessary to extend into the coronal third of the radicular space to reach healthier tissue. Because of the depth at which this procedure is performed, calcium hydroxide should be used as the pulp-capping agent. In the event of failure, the calcium hydroxide allows easy reentry into the canal to perform apexification procedures, whereas removal of MTA would present difficulties. If hemorrhage cannot be controlled in the normal manner used with the Cvek pulpotomy, a compromise should be considered. The hemorrhage may be controlled with hemostatic agents, such as aluminum





**Figure 3-4** Cvek partial pulpotomy technique. **A**, Maxillary central incisor of an 11-year-old girl with complicated crown fracture before treatment with a Cvek partial pulpotomy. **B**, Removal of exposed pulp with high speed to a depth of 2 mm and rinsing till hemostasis. **C**, Sealing the pulp with MTA followed by interim restorative material and glass ionomer liner. **D**, Completed composite restoration. (Courtesy Dr. Hadas Katz-Saghy.)

chloride or ferric sulfate.<sup>40</sup> If successful, the additional root formation will provide additional strength for the tooth. Obviously, this treatment is used only in teeth with blunderbuss apices.

Because it is difficult to determine the status of the pulp deep in the root canal, the outcome may be unpredictable. In the event of nonhealing, traditional apexification procedures should be carried out, which is described later in this chapter.

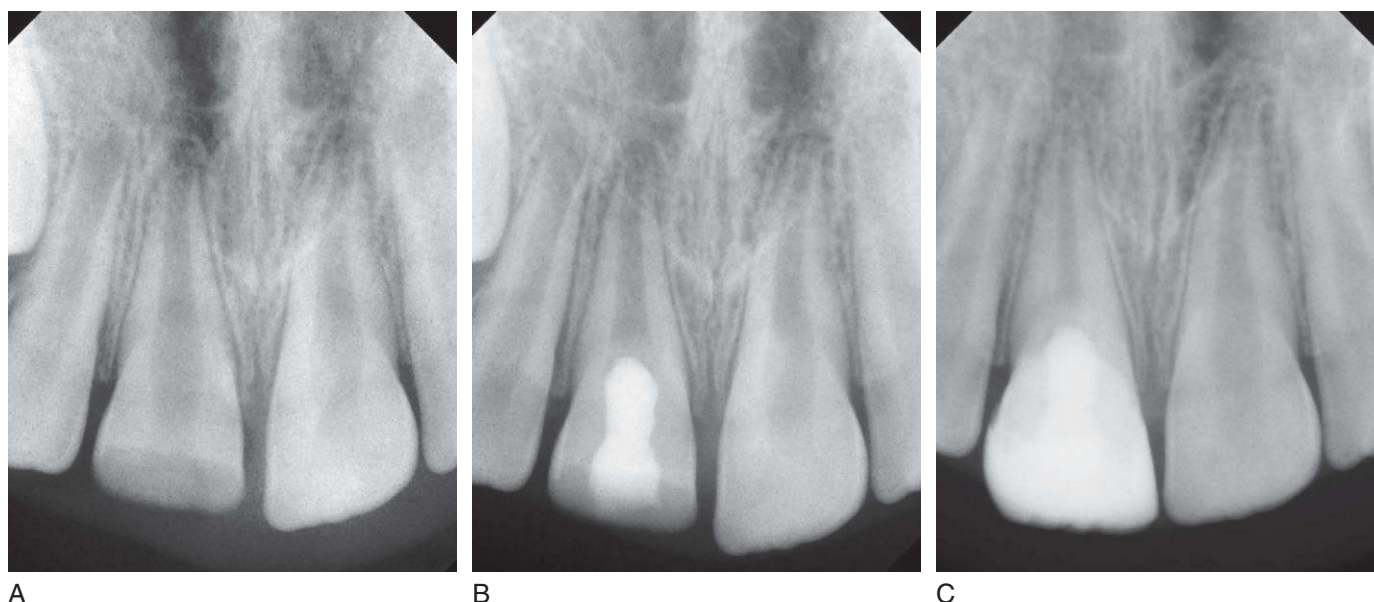
#### Follow-up to Pulp Capping and Pulpotomy

According to Cvek,<sup>40</sup> clinical follow-up of pulp capping and pulpotomy should be performed on a regular basis over at least 3 years and should show the following results to verify success:

- No clinical symptoms
- No radiographic signs of pathosis
- Continued root development in immature teeth
- Formation of a hard tissue barrier
- Level of pulp amputation

#### PULP-CAPPING AGENTS

The characteristics of a pulp-capping material are of importance: it must be biocompatible, nonresorbable, capable of adhering to the dentin, capable of establishing and maintaining a good seal to prevent bacterial contamination, and capable of promoting pulp repair. Ideally the dentin bridge formed after direct pulp capping should be without tunnel



**Figure 3-5** **A**, Maxillary central incisor with open apex, crown fracture, and pulp exposure. **B**, Three month follow-up following deep cervical pulpotomy with MTA and composite restoration. Note dentin bridging. **C**, Eighteen months after pulpotomy showing completed formation of the root.

defects that could allow the penetration of bacteria into the pulp at a later stage.<sup>73</sup>

Various materials have been suggested for capping of exposed pulps or after pulpotomy; at present, the use of calcium hydroxide is still the most frequently recommended.<sup>93</sup> When a paste of calcium hydroxide and water is placed on a pulp wound, it causes superficial liquefaction necrosis. The tissue close to the wound loses its architecture, and coagulation necrosis can be observed beneath it. The coagulated necrotic tissue becomes diffusely calcified, and a hard tissue bridge is subsequently formed.<sup>112,113</sup>

The beneficial effect of calcium hydroxide as a capping material is believed to be a result of its initial low-grade irritation of the injured pulp, allowing a favorable tissue response followed by dentin bridge formation.<sup>93</sup> Calcium ions are released from the capping material, forming inorganic precipitations that have been associated with the mechanism controlling cytological and functional changes in the interacting pulpal cells.<sup>127</sup> The high pH and low solubility of calcium hydroxide prolong its antibacterial effect. However, this material is water soluble and, under leaky restorations, might dissolve and be washed out, leaving an empty space under the filling material. Hard setting calcium hydroxide cements can induce dentin bridge formation, but they do not provide an effective long-term seal against bacteria or their byproducts.

The use of dentin-bonding agents for direct pulp capping (hybridization) has been recommended by some investigators.<sup>67-69,99</sup> The rationale for this recommendation is that hybridization provides an effective, permanent seal against bacterial invasion, allowing healing to occur. Researchers<sup>30</sup>

in a review on pulp capping with dentin adhesive systems reported that self-etching adhesive systems can cause inflammatory reactions, which delays pulpal healing and causes failure of dentin bridging. They stated that vital pulp therapy using acidic agents and adhesive resins seems to be contraindicated. Even when a hard tissue barrier was formed and no clinical symptoms were present, histological examination revealed severe pulpal inflammation or necrosis. When the hard tissue barrier is incomplete and has tunnel defects, leakage under the final restoration will bring bacteria in direct contact with the pulp tissue.<sup>33,91</sup>

In the last decade, a mineral trioxide aggregate, ProRoot MTA™, was introduced for direct pulp capping. MTA is a biocompatible material with an antibacterial effect similar to that of calcium hydroxide and provides a biologically active substrate for cell attachment. This feature makes this material effective in preventing microleakage and improving the treatment outcome. MTA stimulates pulp healing by dentin bridge formation with a minimal inflammatory reaction, as seen in exposed pulps of monkeys.<sup>98</sup> Its dentinogenic effect in short-term capping experiments has also been demonstrated in dogs.<sup>44</sup> One researcher<sup>128</sup> demonstrated in dogs that after direct pulp capping, the underlying pulp tissue was consistently normal, and only at a later stage was some hemorrhage observed in the pulp core. After 2 weeks, the beginning of a hard tissue barrier was found, and reparative dentinogenesis resulted after 3 weeks, associated with a firm fibrodentin matrix. This material seems to be very promising as a direct pulp-capping material and has been approved for this use by the American Dental Association (ADA). A preliminary study in human teeth reports better results with

MTA than with calcium hydroxide.<sup>4</sup> Long-term clinical studies in humans are currently underway.

One drawback with the use of the original MTA as a pulp-capping agent had been its potential discoloration effect because of its gray color. This had been a disadvantage when pulp capping an anterior tooth. To overcome this problem, *white* MTA has recently been introduced. Although the exact composition differences of the two types of MTA have not been reported,<sup>24</sup> the ingredients that alter the color might change the overall properties. However, investigators<sup>64</sup> have shown that both types of MTA act similarly when tissue implants are placed subcutaneously in rats. Another research team<sup>95</sup> observed the dental pulp response to both types of MTA in exposed pulps of dogs. They reported that calcified bridges could be seen 1 week after treatment with both types of MTA, with no major differences between the two treatments. These results were not confirmed by other researchers,<sup>2</sup> who observed better results with the gray MTA in a clinical and histological study.

MTA has comparable biocompatibility and tissue cytotoxicity while producing less inflammation than calcium hydroxide. Also, more dentin bridging in a shorter period of time was observed. Therefore MTA is recommended as the agent of choice for pulp capping and shallow pulpotomy procedures.

## PROGNOSIS

Long-term studies have shown very high success rates of pulp capping and partial pulpotomy with respect to pulp survival.<sup>36,39,40,46,47</sup> Radiographic evidence of hard tissue closure of the perforation can be seen 3 months after pulp capping.

The primary factor for pulp survival after a crown fracture is the presence of a compromised pulp circulation secondary to a luxation injury. Crown fracture with a concomitant luxation injury has been shown to have an increased incidence of pulpal necrosis.<sup>6,106</sup> However, teeth with an immature root development have a greater potential for pulp survival.

Cvek and coworkers<sup>36,37</sup> have reported 96% success with pulp capping and pulpotomy using calcium hydroxide on traumatically exposed permanent pulps. Size of the exposure or time between injury and treatment was not critical as long as the superficially inflamed pulp tissue was removed before pulp capping. These studies included both mature and immature roots. Subsequent investigations<sup>46,58</sup> have verified these findings.

Teeth with partial pulpotomies that presented as clinically healed showed no major pathological changes when these pulps were histologically examined.<sup>37</sup> In a long-term follow-up study<sup>39</sup> of partial pulpotomies in permanent teeth, those judged to be healed at 3 years remained healed 10 to 15 years later.

## TREATMENT TECHNIQUES FOR NONVITAL TEETH

### MATURE TEETH

Fractured teeth that are nonvital but have mature apices can easily be successfully treated with conventional endodontic therapy. The prognosis for these teeth is excellent.

### IMMATURE TEETH

#### Apexification

Loss of pulpal vitality in teeth with incomplete root development presents a more complicated treatment with a reduced prognosis because conventional root canal procedures cannot be used for these teeth with open apices. Additionally the loss of pulpal vitality results in the cessation of root development, leaving weaker roots that are much more prone to fracture than fully developed roots. Lack of complete root development may also result in a poor crown-root ratio, leaving the teeth more susceptible to periodontal involvement because of excessive mobility. Therefore treatment should be directed toward the preservation of pulpal tissue whenever possible to allow for the completion of root formation. Apexification is a method of treatment for immature permanent teeth in which root growth and development ceased because of pulp necrosis. Its purpose is to allow the formation of an apical barrier, and it is most often performed on incisors that have lost vitality subsequent to a traumatic injury.<sup>40,49</sup> Before deciding on this mode of treatment, a correct diagnosis should be determined based on clinical and radiographic assessment. It may also be indicated in nonvital immature teeth after carious exposures and in certain anatomical variations, such as dens invaginatus.

The apex of immature teeth may present in two variations: divergent and flaring apical foramen (blunderbuss apex) or parallel to convergent. In both forms, conventional endodontic treatment cannot be performed because it is difficult, if not impossible, to achieve an apical seal when conventionally obturated. During apexification, healing of the bone will gradually be observed. Apexification is a predictable procedure, and an apical barrier will be formed in 74% to 100% of cases.<sup>111</sup> The most common complication is cervical crown or root fracture because the cervical portion of the tooth is very thin and may fracture easily.<sup>38</sup>

Apexification is traditionally performed using a calcium hydroxide dressing that disinfects the root canal and induces apical closure. The high pH and low solubility of calcium hydroxide keep its antimicrobial effect for a long period of time; Siqueira and Lopes discussed the mechanisms of its antimicrobial activity in detail.<sup>119</sup> Calcium hydroxide assists in the débridement of the root canal because it increases the dissolution of necrotic tissue when used alone or in combination with sodium hypochlorite.<sup>57</sup> Apexification



requires multiple visits and may take a year or more<sup>74</sup> to achieve a complete apical barrier that would allow for conventional root canal obturation. The time necessary for apexification depends on the stage of root development and the status of the periapical tissue.<sup>49</sup>

### Apexification: Technique

Coronal access should be wide enough to include the pulp horns so as to prevent future contamination and discoloration. Gates-Glidden drills are used to remove the lingual eminence in the cervical portion of the root canal, facilitating instrumentation of all aspects of the canal. Débridement of the root canal, including irrigations with disinfecting solutions like 0.5% to 2.5% sodium hypochlorite or 0.2% chlorhexidine solution followed by sterile saline solution, should be done without pressure, verifying that the needle is loose inside the root canal. Minimal instrumentation is advised to prevent damage to the thin dentin walls. A calcium hydroxide dressing in a paste consistency is inserted into the canal. This can be accomplished either with a lentulo spiral mounted in a low-speed engine, with specially designed syringes, or with files. This paste is then compacted with endodontic pluggers. The second appointment should be 2 weeks to 1 month later. The aim of this visit is to complete the débridement and remove any tissue remnants denatured by the calcium hydroxide dressing that could not be removed mechanically during the first appointment. The length of the root canal should be determined radiographically because electronic apex locators are not reliable in teeth with open apices.<sup>65</sup> At the second visit, a thick paste of calcium hydroxide will be packed in the canal space using endodontic pluggers.<sup>85</sup>

The tooth is monitored clinically and radiographically at 3-month intervals. When a calcified barrier can be seen on the radiograph, the tooth is reopened and biomechanically cleaned by sodium hypochlorite irrigation. The apical area should be examined using a file or the blunt end of a thick paper point to the working length to determine the completeness of the apical barrier. If the barrier is incomplete or the patient feels the touch of the file, the apexification procedure is repeated until a complete barrier is formed. The frequency with which the calcium hydroxide dressing should be changed is controversial. Some authors believe that frequent replacements can increase the speed of barrier detection, but does not appear to affect its position.<sup>72</sup> Other investigators<sup>27</sup> have demonstrated that changing the calcium hydroxide dressing did not enhance either the speed nor the quality of the apical barrier in monkeys. Figure 3-6 shows an immature maxillary central incisor with a necrotic pulp and associated sinus tract. The canal was treated with a calcium hydroxide apexification. An apical barrier was achieved, and the bone lesion healed; endodontic treatment was subsequently completed.

When a calcified barrier is formed coronal to the apex, it should not be perforated to fill the tooth to the apical end; the tissue forming the apical barrier should be regarded as

healthy tissue. It has been reported that calcium hydroxide may greatly increase the risk of root fracture after long-term application. Therefore to reduce this risk in immature teeth, it is advisable to minimize the time necessary for apexification.<sup>15</sup>

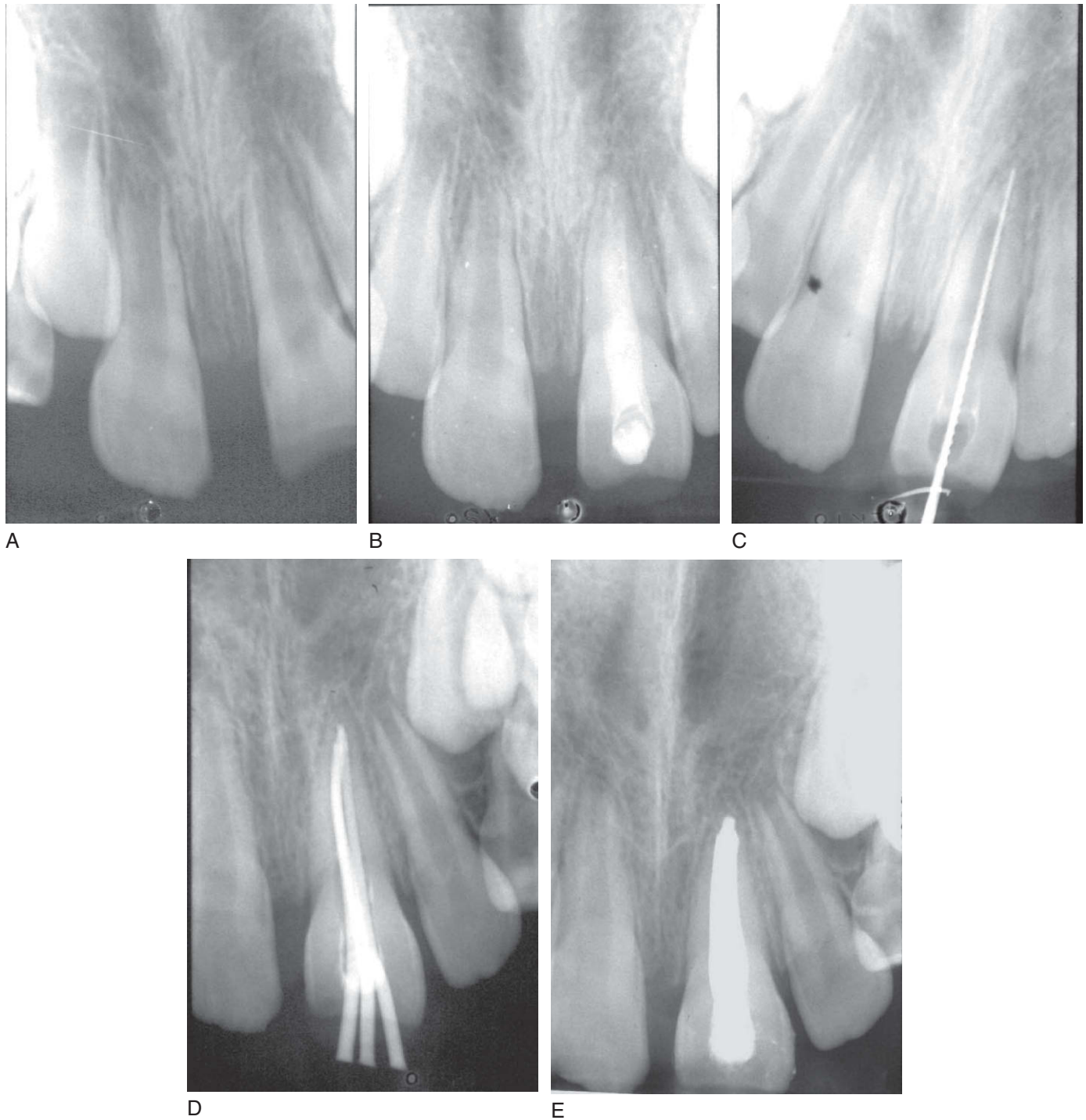
### Apical Barrier: Technique

Although apexification procedures with calcium hydroxide and other materials have been highly successful, many problems exist. During the long treatment period (12 to 18 months), the weak root is left vulnerable to fracture. Patient compliance may be difficult in some cases because of the time factor and number of appointments. With the development of an apical barrier technique using MTA, along with root strengthening restorations of bonded composite, a more predictable outcome is now possible.

Since first reported in 1996,<sup>123</sup> the use of MTA as an apical barrier is becoming the standard for treatment of immature roots with necrotic pulps. When compared with osteogenic protein-1 and calcium hydroxide, MTA induces apical hard tissue more often while producing less inflammation. MTA is less cytotoxic than other commonly used reverse fill materials, such as amalgam, IRM™ (Caulk/Dentsply, Milford, Del.) or Super EBA™ (Bosworth, Skokie, Ill.).<sup>70,94,111,124</sup> Studies<sup>83,126</sup> have shown MTA to be biocompatible; it produces direct bony opposition on implanted specimens. When used as a root-end filling, a complete layer of cementum with total closure of the apical foramen is observed.<sup>62,125</sup> MTA, a fine hydrophilic particle, when hydrated produces a colloidal gel with a pH of 12.5, which solidifies into a hard structure in 4 hours.<sup>126</sup> Investigators<sup>108</sup> stored endodontically treated teeth filled with MTA for 2 months in a synthetic tissue fluid composed of a neutral phosphate buffer saline solution and the dentin of extracted teeth. Interaction with the fluid produced an adherent precipitate on the dentinal wall. This precipitate had the same composition and structure as hydroxyapatite. It was concluded that phosphate in the tissue fluid reacts with calcium released from the MTA, producing hydroxyapatite, and that the biocompatibility, sealing ability, and dentinogenic activity occur because of these physiochemical reactions.

Other research<sup>78</sup> has shown that the physiological environmental effects on MTA are partially determined by pH and the presence of ions. The physical properties, hydration, and microhardness are adversely affected by an acidic environment as would be found with infection and suppuration. Thus these issues should be cleared up before the placement of MTA.

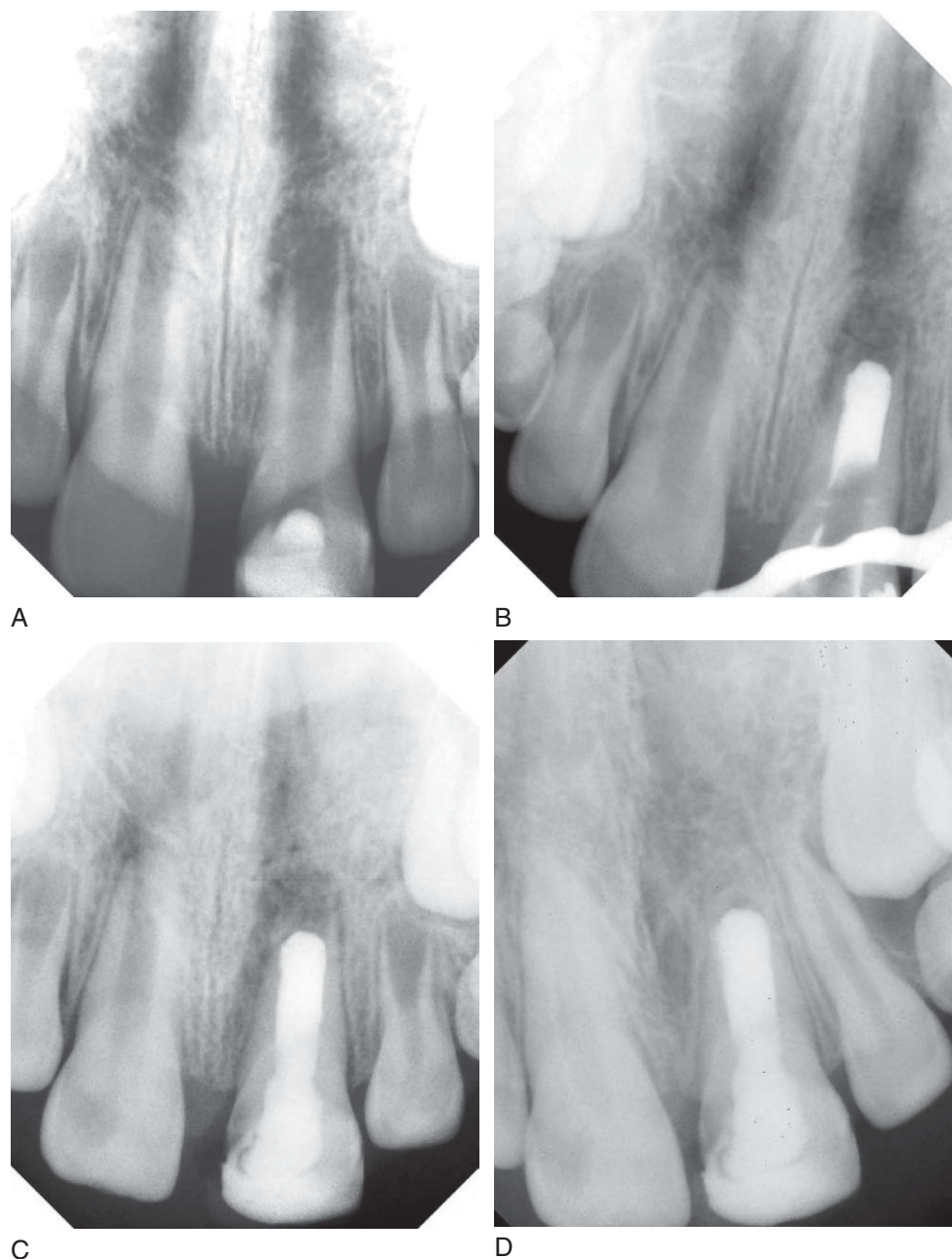
The treatment using an apical barrier with MTA is as follows. The tooth is isolated with a rubber dam, and the canal is cleansed as in any root canal procedure (Fig. 3-7, A). The use of sonic or ultrasonic endodontic devices is helpful in cleaning and flushing the apically divergent canal. Frequent irrigation with sodium hypochlorite helps remove debris. After thorough débridement, the canal is then dried and medicated with a slurry of calcium hydroxide and tem-



**Figure 3-6** **A**, An immature fractured central incisor with pulp necrosis after trauma. **B**, The tooth was débrided and apexification with calcium hydroxide was started. **C**, Six months later the presence of an apical barrier was observed and the calcium hydroxide was removed to place a root filling. **D**, Gutta-percha master point followed by lateral compaction technique. **E**, Completed root filling. (Courtesy Dr. Nathan Rozenfarb.)

porarily sealed to achieve disinfection. After the tooth is free of signs and symptoms of infection, it is again isolated with the rubber dam. It is usually not necessary to anesthetize the patient at this appointment. The canal is reentered and thoroughly cleansed of the medicament and dried. A plug of

MTA is then placed apically. The MTA is mixed as thick as possible and picked up in an amalgam carrier. The pellet of MTA is extruded into the access cavity and gently teased to the apex with the reverse end of a large paper point, which has been premeasured to be 1 to 2 mm short of the working



**Figure 3-7** Apical barrier technique with MTA and bonded composite strengthening root in maxillary central incisor. **A**, Incompletely developed root with open apex and periapical radiolucency. **B**, Apical barrier of MTA. **C**, Bonded composite to strengthen the root. **D**, Three years after treatment showing healing of lesion and closure of apex with cementum.

length. Additional increments of MTA are compacted to the apex until a plug 4 to 5 mm thick is achieved. As the MTA is compacted, the paper point is slightly shortened. This technique, if used carefully, will provide a dense layer of MTA while preventing apical extrusion. Additionally, large, blunt-ended pluggers may be useful in compacting the MTA. Assessment of the adequacy of the apical plug is verified radiographically (Fig. 3-7, *B*).

A dye leakage study<sup>129</sup> comparing different thicknesses of MTA in root end-filled teeth showed that a 4 mm thickness

of MTA was significantly more effective than lesser amounts in preventing dye penetration. The authors suggested a thickness of 4 mm in root end-fillings. Other researchers<sup>3</sup> studying bacterial penetration in simulated open apex teeth with MTA plugs showed that a 5 mm depth completely prevented bacterial leakage, whereas 2 mm failed to do so.

Once the apical plug is satisfactorily placed, the dentinal walls above the MTA are cleansed of all excess material. The walls should be scrubbed with damp paper points or small cotton-tipped plastic applicators in preparation for place-



ment of a bonded composite resin to strengthen the root. All MTA is removed coronal to the apical plug to achieve a dentin-bonded resin interface for maximum strength.

A very wet cotton pellet is then placed in the canal, but should not contact the MTA, or the fibers will adhere to the material when it sets. Dry cotton pellets are placed above the wet pellet and the access is sealed with Cavit™ (ESPE, Seefeld, Germany).

At a subsequent appointment at least 6 hours later, the tooth is reisolated, and the Cavit™ and cotton are removed. Verification of set of the MTA is performed with a probe or endodontic file. A bonded composite resin is then placed in the canal. There are also reports that suggest it may be possible to promote the regeneration of pulp tissue in non-vital immature teeth, which is elaborated on in Chapter 6.

## RESTORATION OF IMMATURE ROOTS

Although apexification procedures have traditionally had a high degree of success, many of these teeth are lost because of the fracture of the thin roots, both during the extended treatment or following completion. Placement of an acid etched, bonded composite resin in the roots has virtually eliminated these fractures. Newer dentinal bonding techniques have been shown<sup>60,69</sup> to strengthen endodontically treated teeth to levels similar to those of intact teeth.<sup>69</sup> When compared with gutta-percha alone,<sup>53</sup> resin-modified glass ionomer with a translucent curing post also showed significantly increased resistance to root fracture in immature roots. A more recent study<sup>77</sup> reported significantly greater resistance to root fracture following placement of a 4 mm apical plug of MTA and intracanal bonded composite resin compared with MTA and gutta-percha and sealer.

After verification of the setting of the MTA apical plug, the dentin is acid etched and a dentinal bonding agent is applied to the internal surfaces of the root directly over the MTA. Following curing of the dentin bonding agent, 2 mm increments of a compactable light cure composite resin, such as Esthet-X™ (Caulk/Dentsply, Milford, Del.) or Alert™ (Pentron Clinical Technologies LLC, Wellington, Conn.) are placed in the canal and thoroughly light cured. To assure curing of the composite in the canal, the curing light is applied at 4 times the normal exposure time.

If a post is necessary to retain a crown because of extensive crown fracture, the Luminex™ clear plastic post (Dentatus USA, New York, N.Y.) without serrations can be used to create post space. The canal is acid etched, and the bonding agent applied and cured. The canal is then filled with a flowable light cure composite resin, being careful to avoid trapping bubbles. The appropriate Luminex™ post is placed to the depth of the preparation in the center of the canal and the resin is cured by transmitting light through the post. The Luminex post is gently removed and a correspon-

ding Dentatus metal post is cemented into the space. The metal core provides a scaffolding on which a core can be fabricated with composite resin.<sup>69</sup>

## CROWN FRACTURES IN PRIMARY TEETH

### CLINICAL APPEARANCE

The clinical appearance of crown fractures in primary teeth is similar to that of permanent teeth. If an enamel infraction is observed, a control radiograph should be exposed after 6 weeks.

### RADIOGRAPHIC APPEARANCE

Findings are similar to the permanent dentition; however, it is important to determine the proximity of the primary tooth in relationship to the developing follicle. This is particularly true in cases of oral luxation and intrusion because the root of the primary tooth can damage the developing permanent successor.<sup>14</sup>

### BIOLOGICAL CONSIDERATIONS

Animal studies have demonstrated that pulpal and periodontal healing responses to acute dental trauma are similar in the primary and permanent dentitions.<sup>11,13</sup>

### UNCOMPLICATED CROWN FRACTURES (NO PULP EXPOSURE)

*Small* uncomplicated crown fractures (enamel only): smoothing or rounding of the sharp edges is usually satisfactory treatment.

*Large* uncomplicated crown fractures (enamel and dentin): treated similar to the permanent dentition. If the fracture is extensive, whereby exposing a considerable amount of dentinal tubules are exposed, a composite strip crown is indicated.<sup>75</sup>

### COMPLICATED CROWN FRACTURES (PULP EXPOSURE)

#### Incidence

Complicated crown fractures are only 1% to 3% of all injuries in the primary dentition.<sup>56,76</sup>

#### Treatment Options

There is no consensus among clinicians on how to treat a complicated crown fracture in the primary dentition, and individual preferences exist.<sup>87</sup> Traditionally the treatment options consist of direct pulp capping, pulpotomy

(partial/coronal), pulpectomy, or extraction.<sup>45,137</sup> Although some recommend treating exposures in primary teeth with pulpotomy or pulpectomy,<sup>52,71</sup> others will select a formocresol pulpotomy as the treatment of choice.<sup>101</sup> If the child is uncooperative, extraction has also been suggested.<sup>14</sup>

**Direct Pulp Capping: Technique** Some practitioners prefer pulp capping when the exposure is very small and when it can be treated immediately after the injury.<sup>76</sup>

**Partial Pulpotomy or Cvek Technique** This technique is widely used in permanent teeth with a high percentage of success,<sup>76,100</sup> but only recently has this treatment modality been recommended for primary teeth. It is important that sufficient tooth structure is present to allow proper restoration and full coverage with a resin-bonded strip crown. Similar to permanent teeth, success is increased if the exposure site and the surrounding dentin are properly sealed from oral contaminants, fluids, and bacteria.<sup>31</sup>

The clinical procedure is similar to that described above for the permanent dentition (Fig. 3-8). However, in very young patients, proper emotional and psychological patient management should be achieved, with or without premedication. After completion of the partial pulpotomy, the tooth is restored with a bonded resin-composite strip crown, as described above.

**Pulpotomy (Partial/Coronal)** When the pulp exposure is very large or when more than 2 weeks have passed since the injury, contaminants may cause extensive infection or inflammation 2 to 3 mm beyond the exposure. In these cases, a coronal pulpotomy may be indicated. A case of a deep subgingival fracture treated with a formocresol pulpotomy is illustrated in Figure 3-9.

A major controversial issue regarding pulpotomy in primary incisors is the question of which agent to use when a primary pulp is treated. Formocresol has been the medication of choice for many years and its use is still widespread, mainly because of its high success rate.<sup>122</sup> Calcium hydroxide may promote internal root resorption in primary teeth.<sup>101</sup> Formocresol presumably fixes affected and/or radicular tissue so that a chronic inflammation replaces an acute inflammation.<sup>101</sup>

Because of the topical and systemic toxicity of formocresol and the assumption that calcium hydroxide causes internal resorption in primary teeth, other alternative agents and techniques have been suggested and studied.<sup>50,79</sup> Numerous agents and techniques have been suggested, including ferric sulfate, glutaraldehyde, bone morphogenetic proteins, electrocautery, and lasers for pulp amputation.<sup>50,79,99</sup>

It is beyond the scope of this chapter to elaborate on these newer and still experimental techniques, but the reader is encouraged to look for future developments in the field.

It is important to emphasize that the key to success, regardless of the clinician's choice of agents, is the prevention of marginal microleakage of the restoration, which could lead to bacterial contamination.

**Pulpectomy** This treatment modality should be considered when a traumatized primary incisor displays chronic inflammation or necrosis in the radicular pulp. The main purpose of retaining the tooth by endodontic therapy is to maintain esthetics and function. Extraction of the involved incisor may result in space loss, unless space is maintained with an appropriate appliance.<sup>76</sup>

The ideal root canal paste for pulpectomies in primary teeth should be able to be absorbed at a pace similar to the physiological resorption of the primary root. If the material is expressed beyond the apex, it should be absorbed easily and be nontoxic to the periapical tissue and succedaneous permanent tooth follicle. Other requirements include the ability of the material to be antiseptic, able to fill the root canal easily, be radiopaque, and not discolor the treated tooth.<sup>90</sup> The most popular root canal filling materials for primary teeth are zinc oxide and eugenol, iodoform paste, and calcium hydroxide.<sup>100</sup> Zinc oxide and eugenol is far from ideal because of its resistance to undergoing absorption.<sup>107</sup> Iodoform pastes have been shown to produce excellent clinical results; they are both absorbable and have long-lasting antibacterial properties.<sup>50,51</sup> Other pastes containing calcium hydroxide and iodoform (Vitapex, NeoDental, International Inc., Bunaby, British Columbia, Canada; Endoflas, Sanlor Laboratories, Cali, Colombia, South America) have demonstrated good clinical results.<sup>51,90</sup>

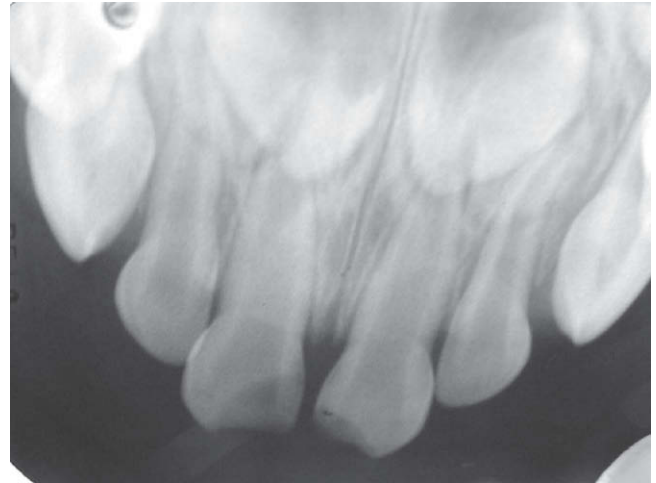
The most frustrating aspect of a pulpectomy is the presence of discoloration in the successfully treated tooth, which may be a cause of parental or child dissatisfaction. The color expected after successful pulpectomy and root canal filling with an absorbable endodontic iodoform paste is yellow. In some cases, the tooth may even appear dark brown. Another disadvantage of root treatment is the possibility of an ectopic eruption of the successor caused by a nonabsorbing paste and the overretention of the primary incisor (Fig. 3-10).

The classic pulpectomy technique has been described in detail in many textbooks.<sup>50,79</sup> A pulpectomy is not recommended when the tooth exhibits extreme loss of coronal tooth structure, advanced internal and/or external resorption, or periapical infection extending to the crypt of the succedaneous tooth.<sup>48,50</sup>

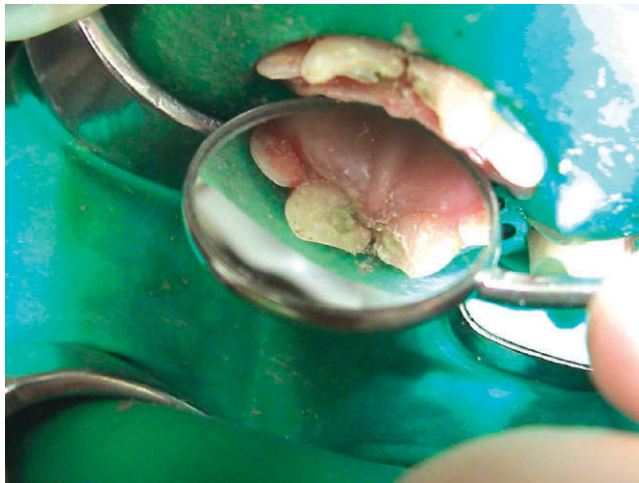
**Extraction** When the previously described treatment options are contraindicated, the treatment of choice is extraction of the affected tooth (Fig. 3-11). The clinician should consider the need for space maintenance following the extraction of primary incisors. Although space maintenance in the posterior region is an important consideration when there is an early loss of a primary molar, the anterior segment appears to be stable from canine to canine—even with the early loss of several incisors—with no net loss of space from canine to canine.<sup>35</sup> The space between the mesial surfaces of the primary cuspids tends to remain the same or increase as the child gets older.<sup>86</sup>



A



B



C



D



E

**Figure 3-8** **A**, Maxillary primary incisors with complicated crown fractures to be treated by Cvek pulpotomy with calcium hydroxide. **B**, The fractured teeth showing complete roots with no periapical pathology. **C**, The treated teeth after pulpotomy followed by calcium hydroxide, IRM, and glass ionomer before restoration. **D**, Completed composite strip crowns before rubber dam removal. **E**, Completed restorations. Notice slight bleeding on the gingiva that disappears after a few hours. (Courtesy Dr. Hadas Katz-Saghy.)





A



C

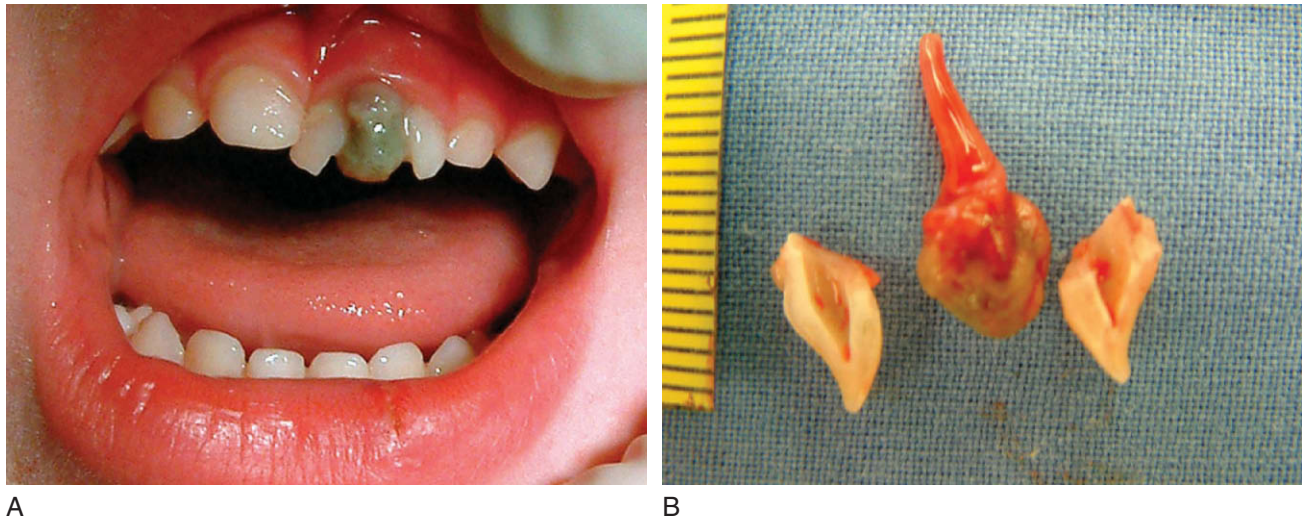


B

**Figure 3-9** **A**, A complicated crown fracture of a maxillary central incisor treated with a formocresol cervical pulpotomy. The palatal fragment was removed and the tooth was restored with a composite strip crown. **B**, The restored tooth 7 months after completing the cervical pulpotomy. Notice the ruptured labial frenum and gingival inflammation. **C**, The treated tooth 7 months after completing the cervical pulpotomy. Notice the composite covering the cervical area.



**Figure 3-10** A retained discolored primary central incisor after root canal treatment with Endoflas. Notice the ectopic eruption of the permanent successor.



**Figure 3-11** **A**, Maxillary left primary incisor with pulp polyp 2 weeks after pulp exposure. The tooth had to be extracted under sedation. **B**, The extracted tooth showing the crown fragments and the pulp from the root. (Courtesy Dr. Elinor Halpersen.)

## REFERENCES

1. Abedi HR, Torabinejad M, Pitt Ford TR et al: The use of mineral trioxide aggregate cement (MTA) as a direct pulp-capping agent, *J Endod* 22:199, 1996 (abstract).
2. Agami HA, Bakry NS, Mounir MM et al: Comparison of mineral trioxide aggregate and formocresol as pulp capping agents in pulpotomized primary teeth, *Pediatr Dent* 26:302, 2004.
3. Al-Kahtani A, Shostad S, Schifferie R et al: In vitro evaluation of microleakage of an orthograde apical plug of mineral trioxide aggregate in permanent teeth with simulated immature apices, *J Endod* 31:117, 2005.
4. Aeinehchi M, Eslami B, Ghanbariha M et al: Mineral trioxide aggregate (MTA) and calcium hydroxide as pulp-capping agents in human teeth: a preliminary report, *Int Endod J* 36:225, 2003.
5. Akimoto N, Momoi Y, Kohno A et al: Biocompatibility of Clearfil linear bond 2 and Clearfil AP-X system non-exposed and exposed primate teeth, *Quintessence Int* 29:177, 1998.
6. Andreasen JO: Luxation of permanent teeth due to trauma. A clinical and radiographic follow-up of 189 injured teeth, *Scand J Dent Res* 78:273, 1970.
7. Andreasen JO, Ravn JJ: Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample, *Int J Oral Surg* 1:235, 1972.
8. Andreasen FM, Andreasen JO: Diagnosis of luxation injuries. The importance of standardized clinical, radiographic and photographic techniques in clinical investigations, *Endod Dent Traumatol* 1:160, 1985.
9. Andreasen FM, Yu Z, Thomsen BL: The relationship between pulpal dimensions and the development of pulp necrosis after luxation injuries in the permanent dentition, *Endod Dent Traumatol* 2:90, 1986.
10. Andreasen FM, Andreasen JO: Crown fractures. In Andreasen JO, Andreasen FM, editors: Textbook and color atlas of traumatic injuries to the teeth, ed 3, Copenhagen, 1994, Munksgaard.
11. Andreasen FM, Andreasen JO: Crown-root fractures. In Andreasen JO, Andreasen FM, editors: Textbook and color atlas of traumatic injuries to the teeth, ed 3, Copenhagen, 1994, Munksgaard.
12. Andreasen FM, Noren JB, Andreasen VO et al: Long-term survival of fragment bonding in the treatment of fractured crowns: a multi-center clinical study, *Quintessence Int* 26:660, 1995.
13. Andreasen JO, Andreasen FM, Bakland LK et al: *Traumatic dental injuries: a manual*, Copenhagen, 1999, Munksgaard.
14. Andreasen JO, Andreasen FM: *Essentials of traumatic injuries to the teeth*, ed 2, Copenhagen, 2000, Munksgaard.
15. Andreasen JO, Farik B, Munksgaard EC: Long-term calcium hydroxide as a root canal dressing may increase risk of root fracture, *Dent Traumatol* 18:134, 2002.
16. Bastone EB, Freher TJ, McNamara JR: Epidemiology of dental trauma: a review of the literature, *Aust Dent J* 45:2-9, 2004.
17. Baume LJ, Holz J: Long term clinical assessment of direct pulp capping, *Int Dent J* 31:251, 1981.
18. Bedi R: The use of porcelain veneers as coronal splints for traumatized anterior teeth in children, *Restorative Dent* 5:55-58, 1989.
19. Bergenholz G, Cox CF, Loesche WJ et al: Bacterial leakage around dental restorations: its effects on the dental pulp, *Oral Pathol* 11:439, 1982.
20. Bernick S, Adelman C: Effect of aging in the human pulp, *J Endod* 1:88, 1975.
21. Bimstein E, Chen S, Fuks AB: Histologic evaluation of the effect of different cutting techniques on pulpotomized teeth, *Am J Dent* 2:151, 1989.
22. Blanco L, Cohen S: Treatment of crown fractures with exposed pulps, *J Calif Dent Assoc* 30:419, 2002.
23. Browning WD, Dennison JB: A survey of failure modes in composite resin restorations, *Oper Dent* 21:160, 1996.
24. Camilleri J, Montesin FE, Brady K et al: The constitution of mineral trioxide aggregate, *Dent Mater* 21:297-303, 2005.
25. Cavalleri G, Zerman N: Traumatic crown fractures in permanent incisors with immature roots: a follow-up study, *Endod Dent Traumatol* 11:294, 1995.

26. Chosack A, Eidelman E: Rehabilitation of a fractured incisor using the patient's natural crown. Case report, *J Dent Child* 7:19, 1964.
27. Chosack A, Sela J, Cleaton-Jones P: A histological and quantitative histomorphometric study of apexification of nonvital permanent incisors of vervet monkeys after repeated root filling with a calcium hydroxide paste, *Endod Dent Traumatol* 13:211, 1997.
28. Ciucchi B, Bouillaguet S, Holz J: Dentinal fluid dynamics in human teeth in vivo, *J Endod* 21:191, 1995.
29. Clark DJ, Sheets GC, Paquette JM: Definitive diagnosis of early enamel and dentin cracks based on microscopic evaluation, *Esthet Restor Dent* 15:391, 2003.
30. Costa CA, Hebling J, Hanks CT: Current status of pulp capping with dentin adhesive systems: a review, *Dent Mater* 16:188, 2000.
31. Cox CF, Keall HJ, Ostro E et al: Biocompatibility of surface sealed dental materials against exposed pulps, *J Prosthet Dent* 57:1, 1987.
32. Cox CF: Microleakage related to restorative procedures, *Proc Finn Dent Soc* 88:81, 1992.
33. Cox CF, Subay RK, Ostro E et al: Tunnel defects in dentin bridges: their formation following direct pulp capping, *Oper Dent* 21:4, 1996.
34. Cox CF, Hafez AA, Akimoto N et al: Biocompatibility of primer, adhesive and resin composite systems on non-exposed and exposed pulps of non-human primate teeth, *Am J Dent* 11:55, 1998.
35. Christensen JR, Fields HW: Space maintenance in the primary dentition. In Pinkham JR, Casamassimo PS, Fields HW et al, editors: *Pediatric dentistry: infancy through adolescence*, ed 4, St Louis, 2005, Elsevier.
36. Cvek M: A clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated crown fractures, *J Endod* 4:232, 1978.
37. Cvek M, Cleaton-Jones P, Austin J et al: Pulp reactions to exposure after experimental crown fractures or grinding in adult monkeys, *J Endod* 8:391, 1982.
38. Cvek M: Prognosis of luxated non-vital maxillary incisors treated with calcium hydroxide and filled with gutta-percha. A retrospective clinical study, *Endod Dent Traumatol* 8:45, 1992.
39. Cvek M: Results after partial pulpotomy in crown fractured teeth 3-15 years after treatment, *Acta Stomatol Croat* 27:167, 1993.
40. Cvek M: Endodontic management of traumatized teeth. In Andreasen JO, Andreasen FM, editors: *Textbook and color atlas of traumatic injuries to the teeth*, ed 3, Copenhagen, 1994, Munksgaard.
41. Delattre JP, Resmond-Richard F, Allanche C et al: Dental injuries among school children aged from 6 to 15, in Rennes (France), *Endod Dent Traumatol* 11:186, 1994.
42. Eid H, White GE: Class IV preparations for fractured anterior teeth restored with composite resin restoration, *J Clin Pediatr Dent* 27:201, 2003.
43. Eidelman E, Holan G, Fuks AB: Mineral trioxide aggregate vs. formocresol in pulpotomized primary molars: a preliminary report, *Pediatr Dent* 23:15, 2001.
44. Faraco IM Jr, Holland R: Response of the pulp of dogs to capping with mineral trioxide aggregate or a calcium hydroxide cement, *Dent Traumatol* 17:163, 2001.
45. Fried I, Erickson P: Anterior tooth trauma in the primary dentition: incidence, classification, treatment methods, and sequelae—a review of the literature, *J Dent Child* 62:256, 1995.
46. Fuks A, Chosack A, Eidelman E: Partial pulpotomy as an alternative treatment for exposed pulps in crown-fractured permanent incisors, *Endod Dent Traumatol* 3:100, 1987.
47. Fuks AB, Gavira S, Chosack A: Long term follow up of traumatized incisors by partial pulpotomy, *Pediatr Dent* 15:334, 1993.
48. Fuks AB: Pulp therapy for the primary and young permanent dentitions, *Dent Clin North Am* 44:571, 2000.
49. Fuks AB, Heling I: Pulp therapy for the young permanent dentition. In Pinkham JR, Casamassimo PS, Fields HW et al, editors: *Pediatric dentistry: infancy through adolescence*, ed 4, St Louis, 2005, Elsevier.
50. Fuks AB: Pulp therapy for the primary dentition. In Pinkham JR, Casamassimo PS, Fields HW et al, editors: *Pediatric dentistry: infancy through adolescence*, ed 4, St Louis, 2005, Elsevier.
51. Garcia-Godoy F: Evaluation of an iodoform paste in root canal therapy for infected primary teeth, *J Dent Child* 54:30, 1987.
52. Garcia-Godoy F, Pulver F: Treatment of trauma to the primary and young permanent dentitions, *Dent Clin North Am* 44:597, 2000.
53. Goldberg F, Kaplan A, Roitman M et al: Reinforcing effect of a resin glass ionomer in the restoration of immature roots in vitro, *Dent Traumatol* 18:70, 2002.
54. Granath LE, Hagman G: Experimental pulpotomy in human bicuspids with reference to cutting technique, *Acta Odontol Scand* 29:155, 1971.
55. Gutz DP: Fractured permanent incisors in a clinic population, *J Dent Child* 38:94, 1971.
56. Hargreaves JA, Cleaton-Jones PE, Roberts GJ et al: Trauma to primary teeth of South African preschool children, *Endod Dent Traumatol* 15:73, 1999.
57. Hasselgren G, Olsson B, Cvek M: Effects of calcium hydroxide and sodium hypochlorite on the dissolution of necrotic porcine muscle tissue, *J Endod* 14:125, 1988.
58. Heide S, Mjor IA: Pulp reactions to experimental exposures in young permanent monkey teeth, *Int Endod J* 16:11, 1983.
59. Heide S, Kerekes K: Delayed partial pulpotomy in permanent incisors of monkeys, *Int Endod J* 19:78, 1986.
60. Hernandez R, Bader S, Boston D et al: Resistance to fracture of endodontically treated premolars restored with new generation dentin bonding systems, *Int Endod J* 27:281, 1994.
61. Holan G, Eidelman E, Fuks A: Long-term evaluation of pulpotomy in primary molars using mineral trioxide aggregate or formocresol, *Pediatr Dent* 27:129, 2005.
62. Holland R, deSouza V, Nery MJ et al: Reaction of dogs' teeth to root canal filling with mineral trioxide aggregate or a glass ionomer sealer, *J Endod* 25:728, 1999.
63. Holland R, de Souza V, Murata SS et al: Healing process of dog dental pulp after pulpotomy and pulp covering with mineral trioxide aggregate or Portland cement, *Braz Dent J* 12:109, 2001.
64. Holland R, de Souza V, Nery MJ et al: Reaction of rat connective tissue to implanted dentin tubes filled with a white mineral trioxide aggregate, *Braz Dent J* 13:23, 2002.
65. Hulsman M, Pieper K: Use of an electronic apex locator in the treatment of teeth with incomplete root formation, *Endod Dent Traumatol* 5:238, 1989.
66. Junn DJ, McMillan P, Bakland LK et al: Quantitative assessment of dentin bridge formation following pulp-capping with mineral trioxide aggregate (MTA), *J Endod* 24:278, 1998 (abstract).
67. Kashiwada T, Takagi M: New restoration and direct pulp capping systems using adhesive composite resin, *Bull Tokyo Med Dent Univ* 38:45, 1991.
68. Kanca J III: Replacement of a fractured incisor fragment over pulpal exposure: a case report, *Quintessence Int* 24:81, 1993.
69. Katebzadeh N, Dalton BC, Trope M: Strengthening immature teeth during and after apexification, *J Endod* 24:256, 1998.



70. Keiser K, Johnson CC, Tipton DA: Cytotoxicity of mineral trioxide aggregate using human periodontal ligament fibroblasts, *J Endod* 26:288, 2000.
71. Kenny DJ, Yacobi R: Management of trauma to the primary dentition, *Ont Dent* 65:27, 1988.
72. Kinirons MJ, Srinivasan V, Welbury RR et al: A study in two centres of variations in the time of apical barrier detection and barrier position in nonvital immature permanent incisors, *Int J Paediatr Dent* 11:447, 2001.
73. Kitasako Y, Murray PE, Tagami J et al: Histomorphometric analysis of dentinal bridge formation and pulpal inflammation, *Quintessence Int* 33:600, 2002.
74. Kleier DJ, Barr ES: A study of endodontically apexified teeth, *Endod Dent Traumatol* 7:112, 1991.
75. Kupietzky A: Bonded resin composite strip crowns for the primary incisors, *Pediatr Dent* 24:145, 2002.
76. Kupietzky A, Holan G: Treatment of crown fractures with pulp exposure in primary incisors, *Pediatr Dent* 25:241, 2003.
77. Lawley GR, Schindler WG, Walker WA III et al: Evaluation of ultrasonically placed MTA and fracture resistance to intracanal composite resin in a model of apexification, *J Endod* 30:167, 2004.
78. Lee YL, Lee BS, Lin FH et al: Effects of physiological environments on the hydration behavior of mineral trioxide aggregate, *Biomaterials* 25:787, 2004.
79. Levine N, Pulver E, Torneck CD: Pulpal therapy in primary and young permanent teeth. In Wei SHV, editor: *Pediatric dentistry: total patient care*, Philadelphia, 1988, Lea & Febiger.
80. Lundy T, Stanley HR: Correlation of pulpal histopathology and clinical symptoms in human teeth subjected to experimental irritation, *Oral Surg Oral Med Oral Pathol* 27:187, 1969.
81. Love RN: Bacterial penetration of the root canal of intact incisor teeth after a simulated traumatic injury, *Endod Dent Traumatol* 12:289, 1996.
82. Matthews B: Sensory physiology: a reaction, *Proc Finn Dent Soc* 8:529, 1992.
83. Mitchell PJ, Pitt-Ford TR, Torabinejad M et al: Osteoblast biocompatibility of mineral trioxide aggregate, *Biomaterials* 20:167, 1999.
84. Mjor IA: Dentin and pulp. In Mjor IA, Fejerskov O, editors: *Histology of the human tooth*, ed 2, Copenhagen, 1979, Munksgaard.
85. Metzger Z, Solomonov M, Mass E: Calcium hydroxide retention in wide root canals with flaring apices, *Dent Traumatol* 17:86, 2001.
86. Moss SJ: Examination, evaluation and behavior following injury to primary incisors. In *Proceedings of the International Conference on Oral Trauma*, sponsored by American Association of Endodontists Endowment & Memorial Foundation, pp 7-15, 1989.
87. Murchison DF, Burke FJT: Partial pulpotomy worth consideration, *Br Dent J* 187:52, 1999.
88. Murray PE, About I, Franquin JC et al: Restorative pulpal and repair responses, *J Am Dent Assoc* 132:1095, 2001.
89. Murray PE, Hafez AA, Smith AJ et al: Bacterial microleakage and pulp inflammation associated with various restorative materials, *Dent Mater* 18:470, 2002.
90. Nurko C, Ranly DM, Garcia-Godoy F et al: Resorption of a calcium hydroxide/iodoform paste (Vitapex) in root canal therapy for primary teeth, *Pediatr Dent* 22:517, 2000.
91. Nyborg H: Healing processes in the pulp on capping, *Acta Odontol Scand* 32:61, 1955.
92. Olgart L, Brannstrom M, Johnson G: Invasion of bacteria into dentinal tubules. Experiments in vivo and in vitro, *Acta Odontol Scand* 32:61, 1974.
93. Olsburg S, Jacoby T, Krejci I: Crown fractures in the permanent dentition: pulpal and restorative considerations, *Dent Traumatol* 18:103, 2002.
94. Osorio RM, Hefti A, Vertucci FJ et al: Cytotoxicity of endodontic materials, *J Endod* 24:91, 1998.
95. Parirokh M, Asgary S, Eghbal MJ et al: A comparative study of white and grey mineral trioxide aggregate as pulp capping agents in dog's teeth, *Dent Traumatol* 21:150, 2005.
96. Pashley DH, Andringa HJ, Derkson GD et al: Regional variability in the permeability of human dentine, *Arch Oral Biol* 32:519, 1987.
97. Pileggi R, Dumsha TC, Myslinksi NR: The reliability of electric pulp test after concussion injury, *Endod Dent Traumatol* 12:16, 1996.
98. Pitt Ford TR, Torabinejad M, Abedi HR et al: Mineral trioxide aggregate as a pulp-capping material, *J Am Dent Assoc* 127:1491, 1996.
99. Prager M: Pulp capping with the total-etch technique, *Dent Econ* 84:78, 1994.
100. Ranly DM, Garcia-Godoy F: Reviewing pulp treatment for primary teeth, *J Am Dent Assoc* 122:83, 1991.
101. Ranly DM, Garcia-Godoy F: Current and potential pulp therapies for primary and young permanent teeth, *J Dent* 28:153, 2000.
102. Ravin JJ: Follow-up study of permanent incisors with enamel cracks as a result of an acute trauma, *Scand J Dent Res* 89:117, 1981.
103. Ravin JJ: Follow-up study of permanent incisors with enamel fractures as a result of an acute trauma, *Scand J Dent Res* 89:213, 1981.
104. Robertson A, Robertson S, Noren JG: A retrospective evaluation of traumatized permanent teeth, *Int J Paediatr Dent* 7:217, 1997.
105. Robertson A, Andreasen FM, Bergenholtz G et al: Pulp reactions to restoration of experimentally induced crown fractures, *J Dent* 26:409, 1998.
106. Robertson A, Andreasen FM, Andreasen JO et al: Long-term prognosis of crown-fractured permanent incisors. The effect of stage of root development and associated luxation injury, *Int J Paediatr Dent* 10:191, 2000.
107. Sadrian R, Coll JA: A long-term follow-up on the retention rate of zinc-oxide eugenol filler after primary tooth pulpectomy, *Pediatr Dent* 15:249, 1993.
108. Sarkar NK, Caicedo R, Ritwik P et al: Physiochemical basis of the biologic properties of mineral trioxide aggregate, *J Endod* 31:97, 2005.
109. Sasafuchi Y, Otsuki M, Inokoshi S et al: The effects on pulp tissue of microleakage in resin composite restoration, *J Med Dent Sci* 46:155, 1999.
110. Schiodt M, Larsen V, Bessermann M: Oral findings in glassblowers, *Community Dent Oral Epidemiol* 8:195, 1980.
111. Sheehy EC, Roberts GJ: Use of calcium hydroxide for apical barrier formation and healing in non-vital immature permanent teeth: a review, *Braz Dent J* 11:183, 1997.
112. Schroder U: Effect of an extra-pulpal blood clot on healing following experimental pulpotomy and capping with calcium hydroxide, *Odontol Rev* 24:257, 1973.
113. Schroder U: A 2-year follow-up of primary molars, pulpotomized with a gentle technique and capped with calcium hydroxide, *Scand J Dent Res* 86:273, 1978.
114. Sheinin A, Pohto M, Luostarinen V: Defense reactions of the pulp with special reference to circulation. An experimental study in rats, *Int Dent J* 17:461, 1967.
115. Simonsen R: Traumatic fracture restoration: an alternative use of the acid etch technique, *Quintessence Int* 2:15, 1979.
116. Simonsen R: Restoration of a fractured central incisor using original tooth fragment, *J Am Dent Assoc* 4:646, 1982.

117. Smales RJ, Gerke DC: Clinical evaluation of four anterior composite resins over five years, *Dent Mater* 8:245, 1992.
118. Smales RJ, Gerke DC: Clinical evaluation of light-cured anterior resin composite over periods of up to 4 years, *Am J Dent* 5:208, 1992.
119. Siqueira JF Jr, Lopes HP: Mechanisms of antimicrobial activity of calcium hydroxide: a critical review, *Int Endod J* 32:361, 1999.
120. Stahlane I, Hedegard B: Traumatized permanent teeth in children aged 7-15 years. Part II, *Swed Dent J* 68:157, 1975.
121. Stanley HR: Management of the aging patient and the aging pulp, *J Calif Dent Assoc* 57:25, 1978.
122. Tagger E, Tagger M: Pulpal and periapical reactions to glutaraldehyde and paraformaldehyde pulpotomy dressing in monkeys, *J Endod* 10:364, 1984.
123. Tittle K, Farley J, Linkhardt M et al: Apical closure induction using bone growth factors and mineral trioxide aggregate, *J Endod* 22:198, 1996 (abstract #41).
124. Torabinejad M, Hong CU, Pitt Ford TR et al: Cytotoxicity of four root end filling materials, *J Endod* 21:489, 1995.
125. Torabinejad M, Pitt Ford TR, McKendry D et al: Histologic assessment of mineral trioxide aggregate as a root-end filling in monkeys, *J Endod* 23:225, 1997.
126. Torabinejad M, Pitt Ford TR, Abedi HR et al: Tissue reaction to implanted root-end filling materials in the tibia and mandible of guinea pigs, *J Endod* 24:468, 1998.
127. Tziafas D: Basic mechanisms of cytodifferentiation and dentinogenesis during dental pulp repair, *J Dev Biol* 39:281, 1995.
128. Tziafas D, Pantelidou O, Alvanou A et al: The dentinogenic effect of mineral trioxide aggregate (MTA) in short-term capping experiments, *Int Endod J* 35:245, 2002.
129. Valois CR, Costa ED Jr: Influence of the thickness of mineral trioxide aggregate on scaling ability of root-end fillings in vitro, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:108, 2004.
130. Vinckier F, Declerck D, Verhaeghe V et al: Injuries to the permanent teeth. Periodontal lesions, *Rev Belge Med Dent* 53:105, 1998.
131. Vojinovic Q, Nyborg H, Brannstrom M: Acid treatment of cavities under resin fillings: bacterial growth in dentinal tubules and pulp reactions, *J Dent Res* 52:1189, 1973.
132. Vongsavan N, Matthews B: The permeability of cat dentine in vivo and in vitro, *Arch Oral Biol* 36:641, 1991.
133. Vongsavan N, Matthews B: Fluid flow through cat dentine in vivo, *Arch Oral Biol* 37:175, 1992.
134. Vongsavan N, Matthews B: Changes in pulpal blood flow and in fluid flow through dentin produced by autonomic and sensory nerve stimulation in the cat, *Proc Finn Dent Soc* 8:491, 1992.
135. Willemsen WL, van der Meer WJ: Repair and revision 4. Cracked tooth and crown fractures: Diagnostics and treatment, *Ned Tijdschr Tandheelkd* 108:170, 2001.
136. Wilson S, Smith GA, Preisch J et al: Epidemiology of dental trauma treated in urban pediatric emergency department, *Pediatr Emerg Care* 13:12, 1997.
137. Wilson CFG: Management of trauma in primary and developing teeth, *Dent Clin North Am* 39:133, 1995.
138. World Health Organization: Application of the international classification of diseases to dentistry and stomatology, ed 3, Geneva, Switzerland: Office of Publishing, World Health Organization, 1995.
139. Zerfowski M, Bremerich A: Facial trauma in children and adolescents, *Clin Oral Invest* 2:120, 1998.

# INTRAALVEOLAR ROOT FRACTURES



LOUIS H. BERMAN

## CHAPTER OUTLINE

### TERMINOLOGY AND EPIDEMIOLOGY

### CLINICAL PRESENTATION

### RADIOGRAPHIC PRESENTATION

### HEALING CLASSIFICATIONS

### SEQUELAE

- Pulp Survival With Canal Obliteration
- Internal Resorption
- External Resorption
- Loss of Crestal Bone

### HEALING PARAMETERS

- Fracture Location
- Diastasis
- Tooth Maturity
- Gender

### MANAGEMENT

- Splinting
- Endodontic Treatment
- Treating the Coronal Fragment
- Treating the Coronal Fragment and Removing the Apical Fragment
- Treating the Coronal and Apical Fragments at the Same Time
- Removing the Coronal Fragment and Treating the Apical Fragment
- Crown-Root Fractures
- Coronal Root Fractures

### PEDIATRIC CONSIDERATIONS

### CONCLUSION

## TERMINOLOGY AND EPIDEMIOLOGY

One of the more unusual sequelae to the traumatic injury of a tooth is a root fracture. There is a wide variety of presentations as to the location, angulation, and severity of these fractures, which provides diagnostic challenges and a myriad of treatment plan options. These fractures may be vertical in relationship to the long axis of the tooth, or they may be more horizontal. When the fracture is more *vertical*, often the tooth is not salvageable or is considered nonrestorable. Extraction may be the only option (Fig. 4-1). These types of fractures typically involve the enamel, dentin, cementum, and often the pulp, and are termed *split roots* or *vertical root fractures*.<sup>1</sup> Unless the tooth is both restorable and periodontally sound, the prognosis is generally poor. When the tooth is fractured such that the root is vertically split, there is currently no predictable method of repair.<sup>9,21,33,45</sup>

However, when the traumatic injury results in a root fracture that is oriented more *horizontally*—and completely encased in bone—often there is a favorable prognosis.<sup>8,13,55</sup> These *intraalveolar root fractures* extend through the entire root—through the cementum, dentin, and pulp<sup>53</sup>—and present as being either *horizontal* (also called *transverse*) or *diagonal* (also called *oblique*). Intraalveolar root fractures may occur at different axial levels of the root and, depending on the location, are classified as:

- Coronal third (Fig. 4-2, A)
- Middle third (Fig. 4-2, B)
- Apical third (Fig. 4-2, C)

The root fracture will produce new sections of the root, which are termed *fragments*. These fragments are designated as being the *coronal* fragment and the *apical* fragment, with the space between these fragments referred to as the *diastasis*<sup>6</sup> (Fig. 4-2, D).

Horizontal intraalveolar root fractures are not very common relative to other types of dental trauma, with an





**Figure 4-1** When a vertical intraalveolar root fracture occurs, it may render the tooth nonsalvageable, and extraction may be necessary, as seen in this maxillary central incisor.

incidence of only 5% to 7%.<sup>8,16,27</sup> These fractures are most commonly seen in maxillary incisors and generally occur in the 10- to 20-year-old age group.<sup>17,55</sup> Male patients are more commonly affected than females.<sup>17</sup> Often there are concomitant associated alveolar fractures.<sup>8</sup> Intraalveolar root fractures seldom occur in primary teeth or in teeth with immature roots.<sup>8,31</sup> Instead, these teeth tend to become luxated within the socket rather than fracturing secondary to a traumatic injury. This is possibly because of the increased elasticity of the alveolar bone surrounding the teeth of these young patients.<sup>31</sup>

## CLINICAL PRESENTATION

Teeth with intraalveolar root fractures tend to have a clinical presentation as being slightly extruded depending on the severity of the dislocation.<sup>5</sup> Most of the time, these injuries are subsequent to trauma directed to the labial aspect of the tooth. This is why the fracture line tends to be more coronal on the labial aspect, extending more apically towards the lingual aspect, with the coronal fragment of the tooth typically being displaced more lingually. Depending on the location of the fracture within the root, there is generally some mobility of the coronal fragment with varying degrees of percussion sensitivity; there may also be some bleeding emanating from the sulcus<sup>5,37,55</sup> (Fig. 4-3). Often the clinical crown will become discolored with varying shades of brown and gray<sup>32</sup> (Fig. 4-4). Possibly owing to the elasticity of pulpal

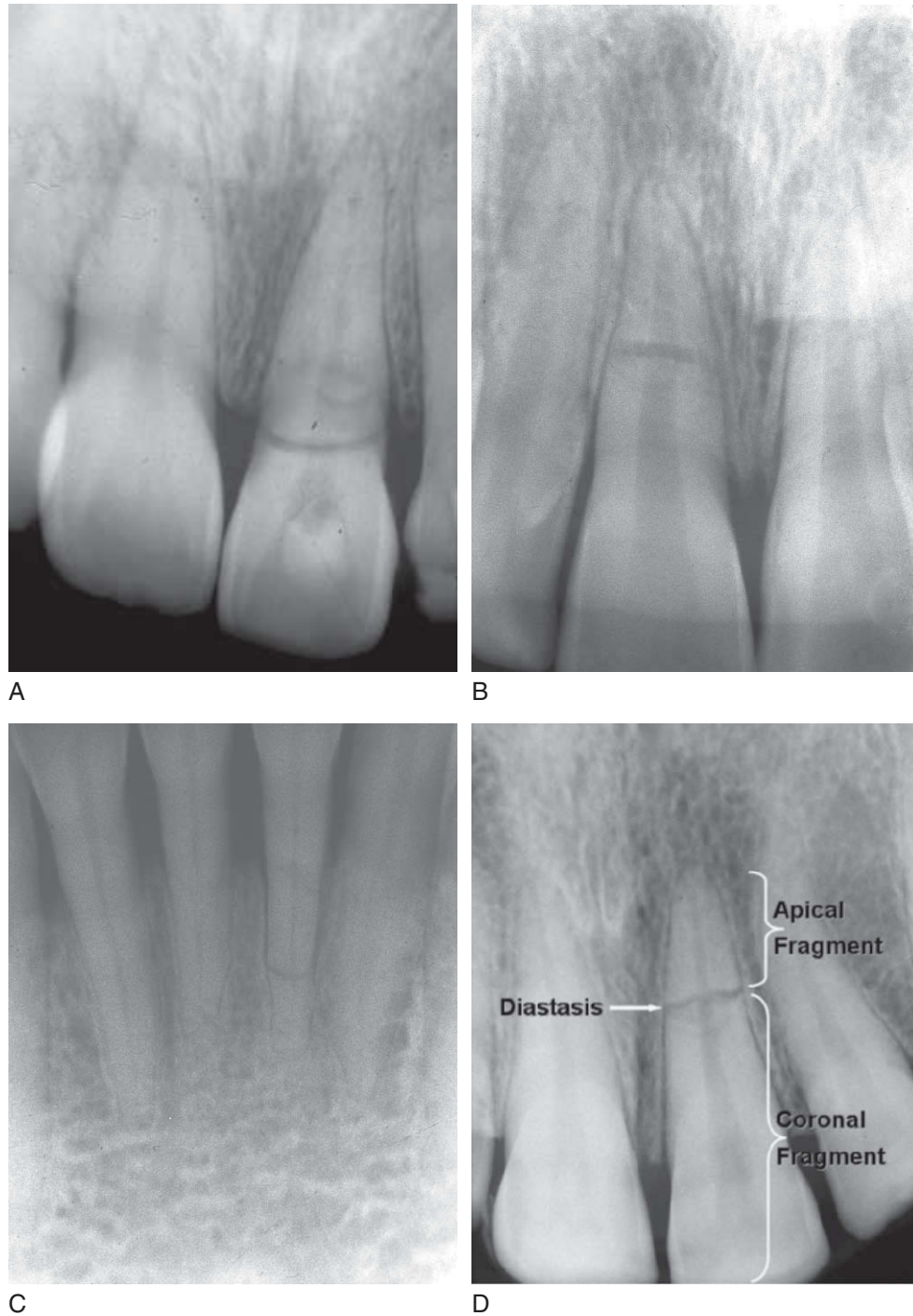
tissue, these teeth have a tendency to remain vital, with many investigators finding that the coronal fragment becomes nonvital only 20% to 24% of the time.<sup>8,10,34,55</sup> In the event that necrosis does occur, it usually happens within the first 2 to 9 months subsequent to the traumatic injury.<sup>32</sup> Interestingly, when the coronal fragment does become nonvital, the apical fragment tends to remain vital.<sup>8,54</sup> Investigators<sup>8</sup> evaluated 12 teeth histologically that had intraalveolar root fractures. They observed that the apical fragments of these teeth remained vital in all 12 cases. This is in contrast to the histological investigation by other investigators<sup>28</sup> whereby one tooth of the six teeth examined showed nonvital tissue in both the apical *and* coronal fragments. The sample sizes of these studies were too small for statistical analysis. But there is enough evidence to suggest that there is a tendency for the apical fragments to remain vital in cases of intraalveolar root fractures.

There is a higher incidence of necrosis among teeth that are traumatized but not fractured.<sup>44,49</sup> There is a presumption that the fracturing of the root may actually reduce the trauma to the pulp by dissipating the energy of the trauma, thus allowing the pulp to remain vital.

## RADIOGRAPHIC PRESENTATION

The radiographic visualization of an intraalveolar root fracture is often difficult. This is why many root fractures are often not diagnosed. When the injury first occurs, the proximity of the fragments may be close, often obscuring the presence of the fracture radiographically. However, after only a few days, the coronal fragment may become more mobile, subsequently increasing its distance from the apical fragment and allowing the fracture to become more visible on a radiograph.<sup>37,55</sup> Also, in the event of nonhealing at the fracture site, bone loss may ensue between the fragments, making the fracture easier to observe. In cervical root fractures where there is communication between the fracture site and the gingival sulcus, there will be a more rapid onset of bone loss and coronal fragment mobility, making the fracture more visible on the radiograph<sup>17</sup> (Fig. 4-5).

When the x-ray beam is directed at approximately the same angulation as the fracture, the fracture will become more visible. This is why the more horizontal the fracture presents (e.g., perpendicular to the long axis of the root), the more likely it is to be visible radiographically. However, most intraalveolar root fractures occur more obliquely to the long axis of the root.<sup>13</sup> Since the x-ray beam is usually directed more horizontally, intraalveolar root fractures are often obscured. Therefore to help facilitate the radiographic detection of a fracture, two or three radiographs should be exposed at various vertical angulations<sup>13,28</sup> (Fig. 4-6, A and D). Also noteworthy is the positioning of the fragments at the fracture site. The fragments are usually oriented such



**Figure 4-2** A, Fracture in the coronal third of the root. B, Fracture in the middle third of the root. C, Fracture in the apical third of the root. D, Illustration of the location of the apical and coronal fragments, and the diastasis.

that they slightly overlap each other. In the two dimensional appearance of the radiograph, this is often deceptively visualized as if a comminuted (e.g., splintered or multiple) fracture is present. Radiographically, this may appear as an elliptical fragment between the coronal and apical fragments<sup>13</sup> (Fig. 4-6, E).

## HEALING CLASSIFICATIONS

The classifications of healing, or lack thereof, are related to the approximation of the coronal and apical fragments, the bony changes that may occur at the fracture site, and the vitality of the pulp tissue within either fragment. On the



**Figure 4-3** Fracture in the coronal third of the root causing sulcular bleeding.

basis of radiographic and histological observations, Andreasen and Hjorting-Hansen<sup>8</sup> have divided the various types of healing from intraalveolar root fractures into four categories:

1. *Calcific fusing of the fragments.* In these cases, there is still radiographic evidence of a fracture line, but there is no radiolucency between the fragments. Instead there is often a radiopaque transition between the fragments, with the appearance of a calluslike repair. Studies have shown that this hard tissue fusion may occur by either pulpal odontoblasts and fibroblasts<sup>8</sup> or solely by cementoblasts.<sup>39</sup> Additionally the canal spaces in the coronal or apical fragments may undergo extensive canal obliteration with calcified tissues,<sup>55</sup> with the pulp tissue usually remaining vital.<sup>13,28</sup> This type of healing occurs about 30% of the time<sup>6</sup> (Fig. 4-7, A).
2. *Connective tissue band between the fragments.* In these cases, there is a radiographic presentation of a narrow radiolucent space between the fragments. The outside edges of the both fragments become rounded adjacent to the fracture site, and the fragments are covered by cementum. The coronal fragment tends to have a wider lamina dura compared with the apical fragment, possibly because of the relative increase in the coronal fragment mobility. This type of healing occurs about 43% of the time<sup>6</sup> (Fig. 4-7, B).
3. *Bone between the fragments.* In these cases, there is a normal and intact lamina dura surrounding each fragment with bone deposition occurring in the area of the diastasis. This type of healing is the most unusual, occurring approximately 5% of the time<sup>6</sup> (Fig. 4-7, C).
4. *Granulation tissue between the fragments (nonhealing).* In these cases, there is typically a wider diastasis with a radiolucency present between the fragments. Inflammation and/or infection in this area may originate either from necrotic pulp tissue or from a communication of the gingival sulcus and the fracture site. This type of presentation is considered nonhealing and occurs about 22% of the time<sup>6</sup> (Fig. 4-7, D).



**Figure 4-4** Fracture in the coronal third of the root causing the clinical crown to become extruded and discolored.

## SEQUELAE

Not only can intraalveolar root fractures have different types of healing, they can also have various types of sequelae, such as:

- Pulp canal obliteration
- Internal resorption
- External resorption
- Loss of crestal bone

## PULP SURVIVAL WITH CANAL OBLITERATION

One of the most frequent findings in an intraalveolar root fracture is canal space calcification. This canal space obliteration, also known as *calcific metamorphosis*,<sup>35,48</sup> typically will be associated with vital pulp tissue, even though the canal space may not be visible radiographically. Therefore barring any other pathological findings, endodontic treatment is not indicated<sup>35,43,48,54</sup> (Fig. 4-8, A).

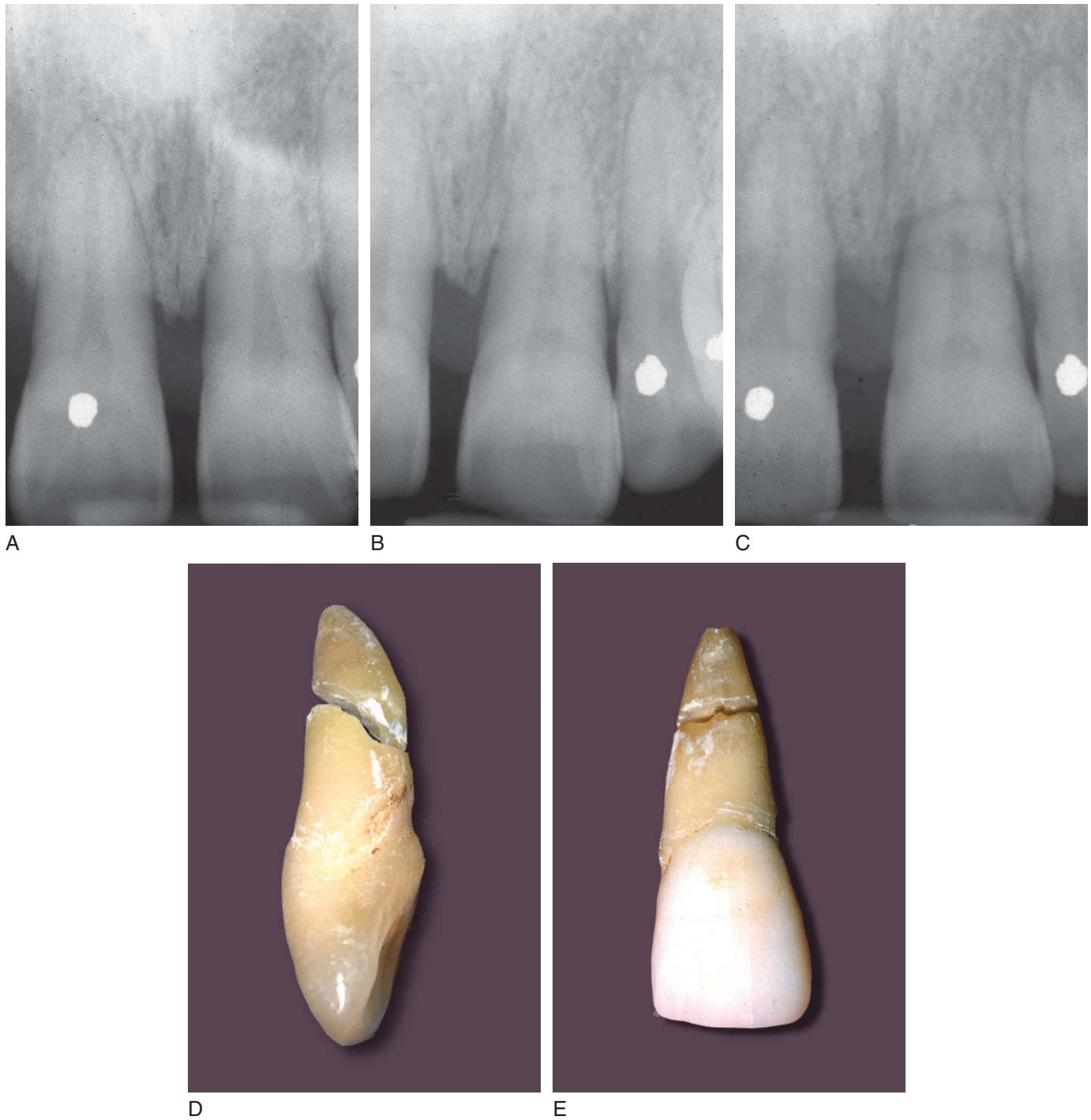
## INTERNAL RESORPTION

Internal resorption is the pathological reaction of the dental pulp as a consequence of a long standing chronic pulp inflammation.<sup>12</sup> Intrapulpal infection has been associated with this resorption, and thought to be a factor in its stimulation.<sup>27</sup> The resorption may be transient, when lacunae are present within the canal walls, or the resorption may become progressive when odontoblasts are destroyed, not permitting predentin to be laid down.<sup>18</sup> Although internal resorption may be a sequela to other traumatic tooth injuries (see Chapters 5 and 6), it is not a frequent finding with intraalveolar root fractures<sup>18,51</sup> (Fig. 4-8, B).

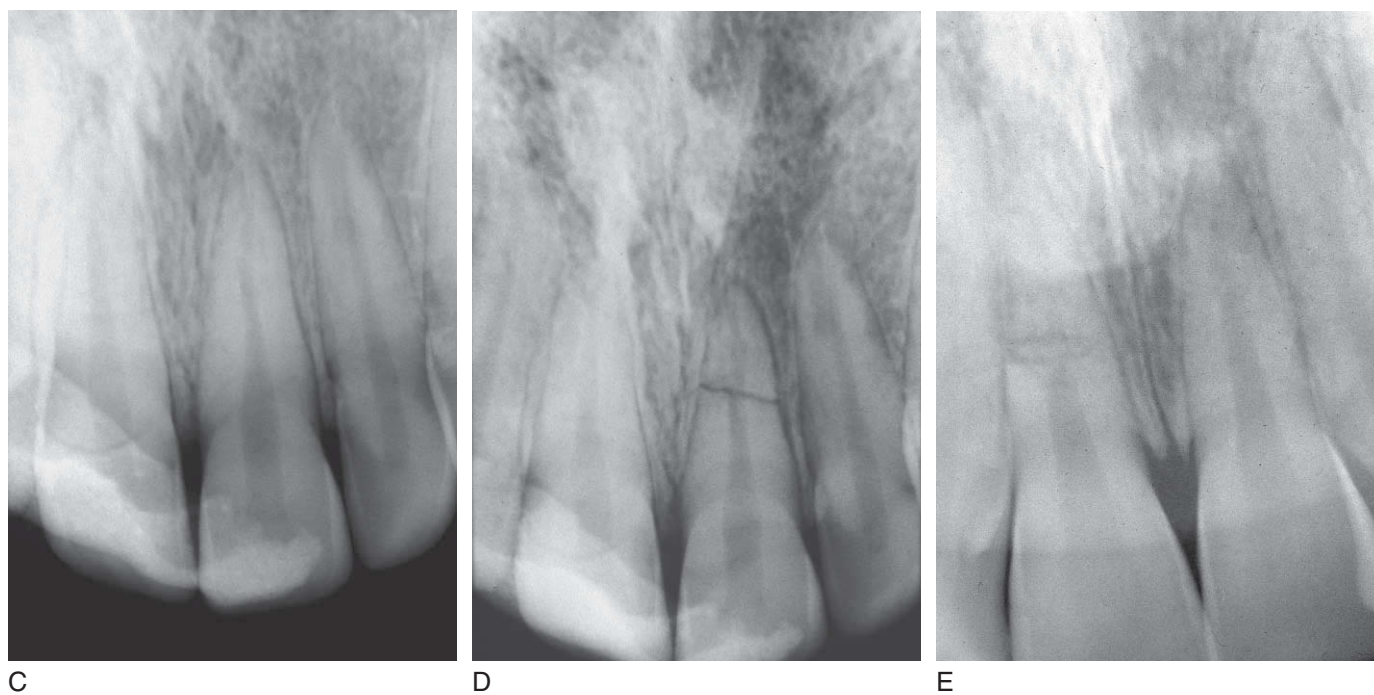
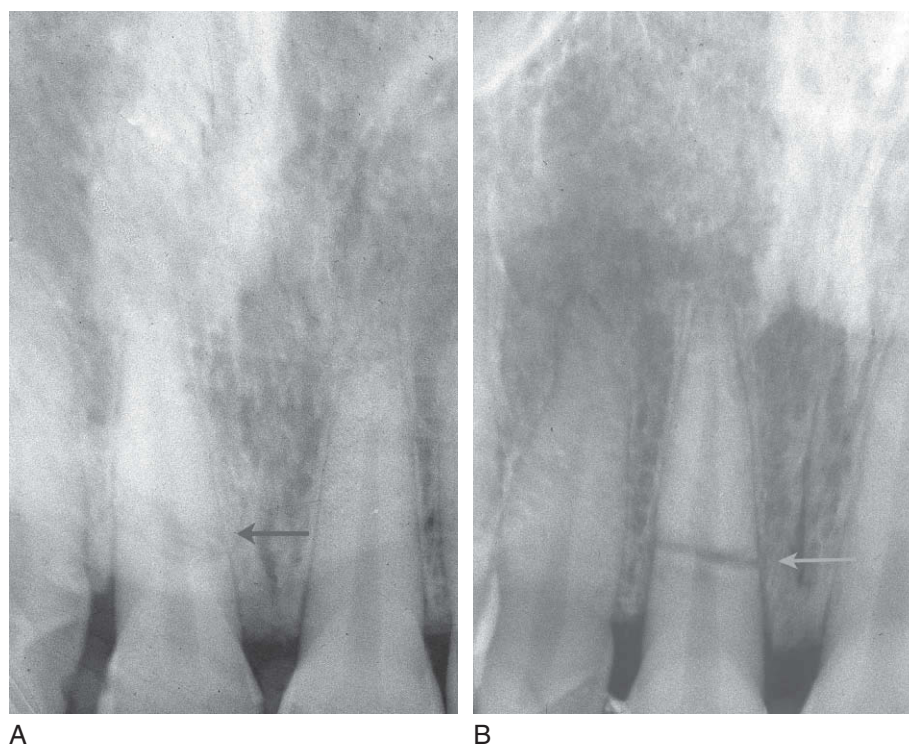
## EXTERNAL RESORPTION

Over time, follow-up radiographs may reveal a resorptive process developing around the areas of the root that are proximal to the fracture site. This is typically a transient, self-

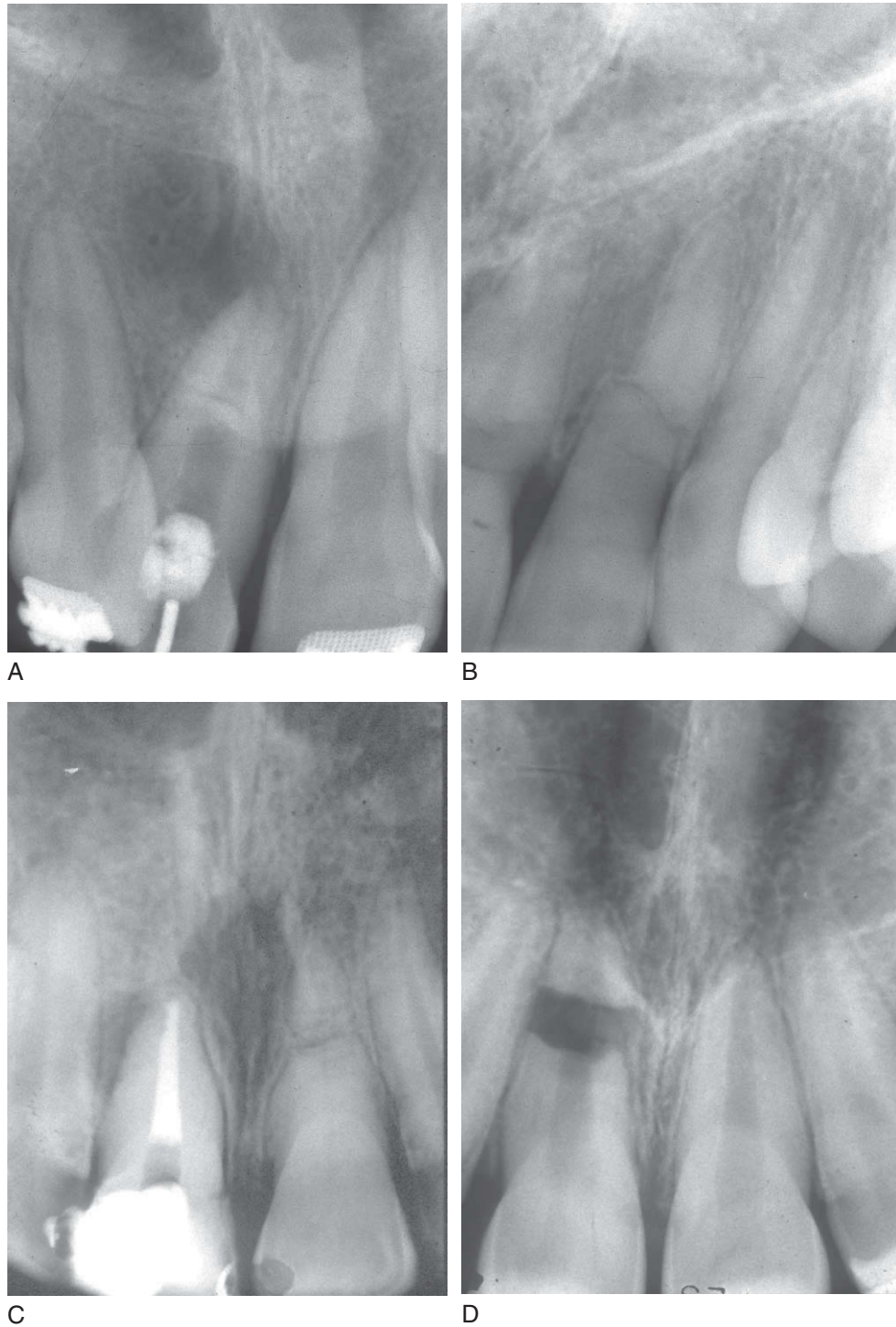




**Figure 4-5** **A-C**, Over time, a fracture in the coronal third of the root may begin to communicate to the sulcus subsequent to periodontal disease. The radiographs depict the bone loss over a period of 3 years. **D** and **E**, Subsequent to the gingival communication, bone loss rapidly ensued, with the result being a severely compromised root. Extraction was ultimately recommended.

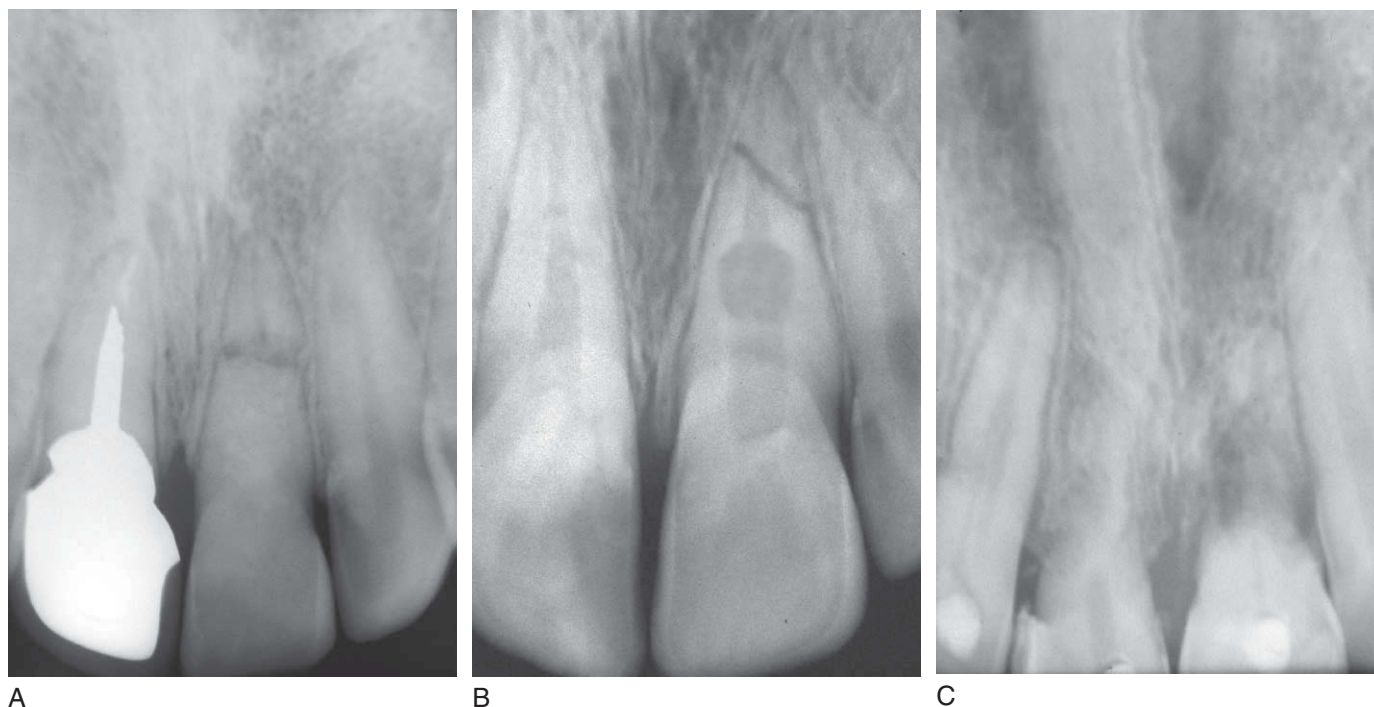


**Figure 4-6** Unless the angle of the x-ray beam is fairly close to the same angulation as the root fracture, the fracture may not be observed. These are radiographs of the same tooth. Part **A** does not adequately exhibit the fracture (*arrow*). **B**, Adjusting the direction of the x-ray beam reveals the root fracture (*arrow*). A similar case is seen in parts **C** and **D**. **E**, Occasionally, because of the overlapping of the proximal fragments at the fracture site, the fracture may appear comminuted, displaying an elliptical appearance on the radiograph.



**Figure 4-7** There are three types of healing of horizontal intraalveolar root fractures, and one type of nonhealing of these fractures. **A**, Illustrates the calcific fusion of the fragments. **B**, Depicts a thin area of connective tissue between the fragments. **C**, Illustrates bone deposition between the fragments. **D**, Depicts nonhealing between the fragments, with a deposition of granulation tissue in the diastasis.





**Figure 4-8** **A**, Calcific metamorphosis of the coronal fragment of the central incisor. **B**, Internal resorption in the coronal fragment of the central incisor. **C**, External resorption associated with an intraalveolar root fracture.

limiting process.<sup>3,29,40</sup> Replacement resorption (i.e., ankylosis, which is the fusing of the root to the surrounding bone—see Chapters 5 and 6) can sometimes be seen in areas surrounding the apical fragment of teeth with intraalveolar root fractures in the apical third<sup>40</sup> (Fig. 4-8, C).

## LOSS OF CRESTAL BONE

Loss of crestal bone is one of the most complicated sequelae of intraalveolar root fractures, especially occurring when there is a coronal root fracture that extends to the gingival sulcus, exposing the fracture site directly to the oral cavity. This generally results in a poor prognosis for the coronal fragment<sup>17,28</sup> (see Fig. 4-5).

## HEALING PARAMETERS

When it comes to intraalveolar root fractures, there are several factors that may increase or decrease the chance of pulp survival and overall healing of the case. These parameters should only be considered general trends and are only predictive of the prognosis. They do not necessarily guarantee success or failure of the case.

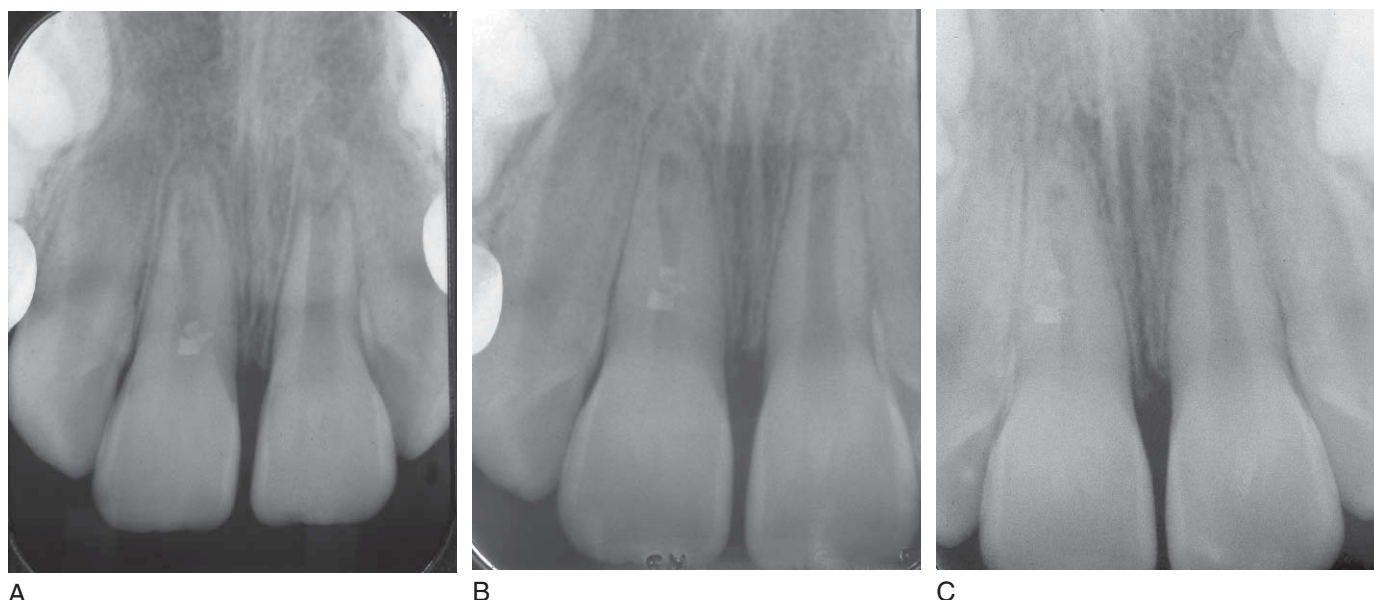
## FRACTURE LOCATION

Generally the more apical the location of the root fracture, the less likely the pulp is to become nonvital.<sup>10,13,19,52,55</sup> In

an investigation comprising 84 teeth with intraalveolar root fractures, coronal third fractures had the worst prognosis, with 61% of them requiring extraction.<sup>42,52</sup> Coronal root fractures may occur in either a *horizontal* or *oblique* orientation. When the fracture is more *oblique*, extending more towards the middle of the root, there is a better chance of healing, possibly because the coronal fragment is more stable within the alveolar bone. Investigators<sup>19</sup> observed this finding when they compared 94 teeth that had fractures in the coronal third of the root. They found that 44% of the teeth had to be extracted when the fracture was more *horizontal* compared with losing only 8% of the teeth when the coronal fracture was more *oblique*. Midroot and apical fractures tend to result in less mobility of the coronal fragment, which is thought to favor better healing.

## DIASTASIS

The space between two normally joined anatomical parts is known as the *diastasis* and is used to describe the space between the fractured fragments of a root.<sup>6</sup> An increase in the diastasis between the root fragments favors necrosis of the coronal fragment, probably because of the increase in pulp tissue damage. A larger diastasis will also tend to increase the mobility of the coronal fragment, which may also decrease the potential for healing of the traumatic injury.<sup>6,20</sup>



**Figure 4-9** The increased vascularity of an immature root may help the tooth stay vital subsequent to an intraalveolar root fracture. This is evident radiographically in **A-C**, each exposed 4 months apart, as the root of the tooth has continued its normal maturation and development.

## TOOTH MATURITY

Young teeth and teeth with immature roots have a greater vascularity than older teeth and are thought to have a better potential for healing after a traumatic injury.<sup>4,25</sup> This has been a consistent finding when evaluating the potential for healing in teeth with intraalveolar root fractures<sup>6</sup> (Fig. 4-9).

## GENDER

In a recent study, girls were seen to have a better potential for healing than boys after injuries that caused intraalveolar root fractures.<sup>6</sup> However, it was noted in this investigation that the girls examined were younger than the boys, and younger teeth tend to heal better after traumatic injuries. Another possible explanation could be that the injuries sustained by the boys may have been the result of a greater impact to the teeth.<sup>6</sup>

Summarizing expected root fracture healing, it depends upon the following factors:

- The location of the fracture, with fractures in the cervical third having the poorest prognosis
- The degree of dislodgment and mobility of the coronal fragment
- The time elapsed between the trauma and the reposition or stabilization of the root fracture
- The tooth maturity
- The status of the pulp tissue
- The loss of crestal bone

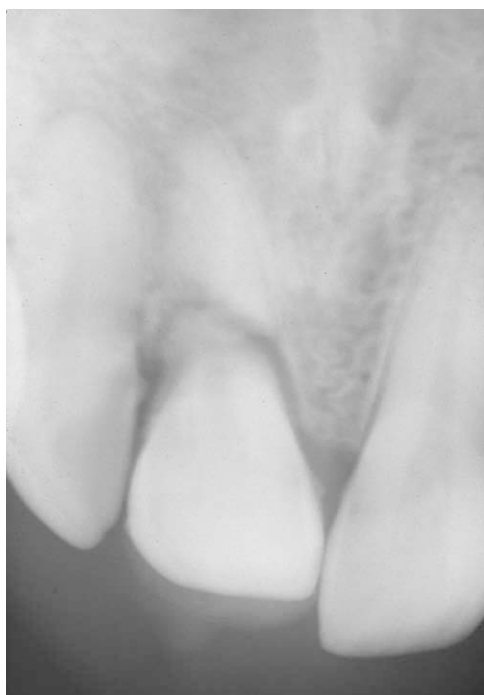
All of the above factors may alter the prognosis of the case. In addition to assessing the case at the time of the injury and

thereafter, the patient should be informed as to how well the case is expected to heal over time.

## MANAGEMENT

Upon the determination that an intraalveolar root fracture is present, there are many issues that must be addressed. The patient should be informed of the problem and given a prediction as to the prognosis with the recommended treatment plan. For example, as previously mentioned, when the fracture is located more apically in the root, the prognosis may be more favorable (24% to 34% success).<sup>17,19,20</sup> Conversely, if the fracture is located in the coronal third, especially if there is extensive mobility of the coronal fragment, then the prognosis is not as favorable (2% to 9% success).<sup>17,19,20</sup> Similarly, there may be a fracture in the coronal third that results in a nonsalvageable coronal fragment, with an apical fragment that has minimal periradicular bone. Given these conditions, the prognosis may be considered poor (43% to 57% success)<sup>17,19,20</sup> and extraction may need to be considered as the treatment of choice (Fig. 4-10).

After assessing the prognosis and advising the patient of the treatment options, proper case management is critical. Generally speaking, similar to a fractured bone, the proper initial management is to reduce the fracture by manually repositioning the coronal fragment back to its proper orientation. When there is excessive mobility of the coronal fragment, proper fixation is essential by splinting the coronal fragment to the adjacent teeth. Because under favorable conditions most of these teeth remain vital, periodic



**Figure 4-10** In the event that there is an intraalveolar root fracture in the coronal third of the root, whereby there is communication to the sulcus, the tooth may be deemed nonsalvageable if the apical fragment is insufficient to support a crown.

radiographic evaluations and pulp vitality tests are essential.<sup>13</sup> In the event that the coronal fragment becomes nonvital, endodontic treatment should be initiated as soon as possible.<sup>13</sup> The specific details of how these cases are managed are described below.

## SPLINTING

It is difficult to generalize as to when and how long it is necessary to splint a case of an intraalveolar root fracture because the location of the fracture, the mobility of the coronal fragment, and the diastasis between the fragments can be so variable. But in a study comparing 400 teeth over 3 years, investigators<sup>7</sup> came to the conclusion that:

- Repositioning the coronal fragment in close proximity to the apical fragment seems to improve healing
- If the fragments are in close proximity subsequent to the trauma, splinting does not make a difference on the healing potential
- Semirigid splinting or no splinting seem to have the most favorable effect on healing
- Splinting for more than 4 weeks does not seem to make a difference on healing
- Delaying treatment for 24 hours does not seem to make a difference on healing

Therefore a splint should be placed only if there is notable mobility or poor positioning of the coronal fragment. If a



A



B

**Figure 4-11** Splinting of the crown to adjacent teeth may be necessary to stabilize the tooth within the arch. Often a monofilament splint is bonded to the loose tooth. Isolation with a rubber dam, if possible, is preferred.

splint is necessary, it should be semirigid and left on for no more than 4 weeks, with probably no benefit beyond that. In the event of a major alveolar bone fracture, splinting for a longer duration may be necessary, as described further in Chapter 7 (Fig. 4-11).

## ENDODONTIC TREATMENT

Since roughly 80% of teeth with intraalveolar root fractures remain vital, endodontic treatment is usually not necessary.<sup>8,13,55</sup> However, assessing pulp vitality in these cases can be difficult. Often, subsequent to any dental trauma, electrical pulp tests may reveal a negative response, with the tooth appearing nonvital; but this may be a false negative.<sup>15</sup> Although not without controversy, if the vitality of the tooth does not recover within 6 to 8 weeks, the coronal fragment should be considered nonvital and endodontic treatment should be initiated.<sup>13,15</sup> Teeth with immature apices may not respond to pulp tests, or they may respond at a very high threshold.<sup>26</sup> A cold test, using carbon dioxide snow, may give a more accurate result.<sup>26</sup> In these cases, where intraalveolar



root fractures are present and the pulp vitality is questionable, the dentist should delay any treatment and follow up with periodic periapical radiographs to assess root development and perform periodic pulp vitality tests.<sup>13,23</sup> The root development over time should be compared with that of the adjacent and contralateral teeth. If the root of the traumatized tooth is not developing, the tooth should be considered nonvital and endodontic treatment should be initiated. Additionally, a tooth that has undergone a traumatic injury may often respond by having a dramatic increase in pulpal calcification over a period of time. Often, these teeth will not respond to pulp tests, even though there is vital pulp tissue present.<sup>13</sup> Therefore, periapical bone loss, bone loss at the fracture site, arrested root development (either apically or in the mesial-distal pulp space dimensions), darkening of the clinical crown, or the development of clinical symptoms should be used as additional aids when determining the existence of pulpal necrosis.<sup>13,17</sup>

Once it has been determined that endodontic treatment is necessary, several options exist\* including:

- Endodontically treat the coronal fragment
- Endodontically treat the coronal fragment and surgically remove the apical fragment
- Endodontically treat both the coronal and apical fragments
- Remove the coronal fragment and endodontically treat the apical fragment

The appropriateness and management of these cases is described below

## TREATING THE CORONAL FRAGMENT

Treating the coronal fragment is the most ideal scenario, and relative to other treatment options, it is technically the most simplistic<sup>20</sup> (Fig. 4-12, A-C). There are two basic problems that may arise when attempting to endodontically treat the coronal fragment in the situation of an intraalveolar root fracture.

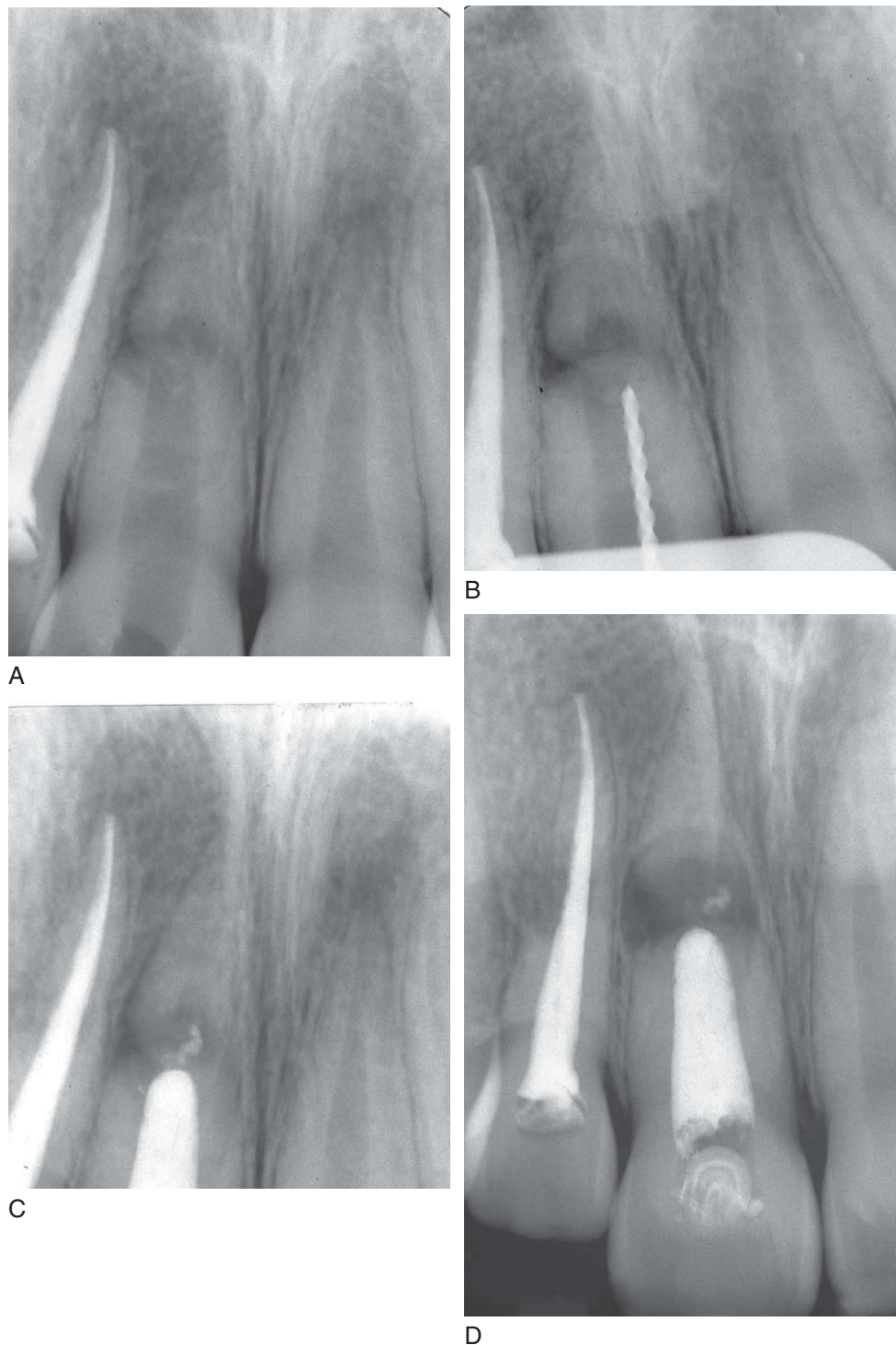
1. If the fracture site is located more coronally on the root, then the “new apex” of the coronal fragment will be “wide” and may be “open,” similar to that of an open apex of an immature root. In the event that the resulting new apical opening of the coronal fragment is significantly wide, then conventional endodontics will become difficult without first performing an apexification procedure.<sup>36,38,41,46,50</sup>
2. Since most fractures are more oblique in nature, the determination of the *location* of the new apical opening of the coronal fragment may be challenging with respect to obtaining an accurate working length measurement. This is especially true if an electronic apex locator is used to determine the working length, since its accuracy is low in cases in which open apices are present.<sup>14</sup>

When properly managed, the success rate of endodontically treating the coronal fragment can be comparable with that of endodontic treatment on a tooth without a root fracture,<sup>55</sup> and *the best option of endodontically managing these cases is to only treat the coronal fragment.*<sup>20</sup> The radiographic estimation of the working length should be measured first. After endodontic access, the size of the apical opening of the coronal fragment should be assessed. If the apical opening of this fragment is considered too wide for conventional endodontic treatment, then an apexification procedure on the coronal fragment should be initiated. Historically, calcium hydroxide has been used, placing it inside the root and replacing it monthly until a hard tissue barrier is felt apically; subsequently the coronal fragment is obturated. Investigators<sup>20</sup> have shown that there may be a 10% greater success rate when the coronal fragment is first treated with a calcium hydroxide apexification before gutta-percha obturation. Conversely the investigators noted that there was a higher incidence of nonhealing when there was an overextension of gutta-percha into the fracture space. Apexification studies are now suggesting that rather than multiple visits using calcium hydroxide applications, equal or better results may be achieved by performing the procedure in one visit using the placement of mineral trioxide aggregate (MTA) apically as an artificial barrier before obturation.<sup>36,38,41,46,50</sup> Similar to calcium hydroxide applications, MTA facilitates the deposition of a hard tissue barrier apically, but has an advantage of allowing the immediate placement of the coronal obturation material. This technique eliminates the necessity of several visits over a course of 3 to 12 months, eliminates potential temporary restoration leakage, and eliminates the potential for interappointment reinfection. After the placement of the MTA plug apically, it has also been suggested to fill the coronal canal space with a dentin bonding material to reinforce the remaining tooth structure.<sup>41</sup> Details of apexification procedures are described in Chapter 3.

## TREATING THE CORONAL FRAGMENT AND REMOVING THE APICAL FRAGMENT

Sometimes after the coronal fragment is endodontically treated, signs or symptoms may suggest nonhealing<sup>5</sup> (see Fig. 4-12, D-H). This may be manifested by swelling or the development of a radiolucent area at the fracture site or apically at the area of the apical fragment. In these cases, assuming that the coronal fragment is stable, surgical intervention is recommended whereby the apical fragment is removed. Subsequent to this, the apical seal of the coronal fragment should be assured with a retrograde restoration. One investigation found the success rate of this type of procedure to be about 68%.<sup>20</sup> Most of the nonhealing cases in this study had to do with an increased mobility or infraposition of the coronal fragment. It has been suggested that when there is notable mobility of the coronal fragment, an implant should be placed through the coronal fragment and into the bone.<sup>5</sup>

\*References 11, 13, 20, 36, 47, 55.

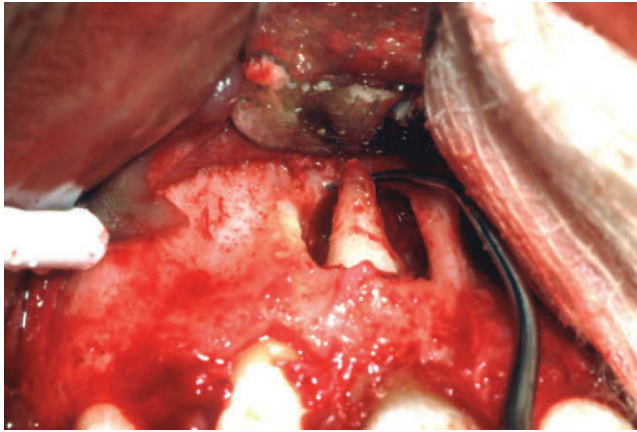


**Figure 4-12** Treatment of the coronal fragment. **A**, Radiograph of how the tooth presented, with an open access. **B**, The working length radiograph. **C**, Shows the obturation of the coronal fragment. Six months after the treatment, swelling ensued, which appeared to originate from the area of the diastasis (**D**).

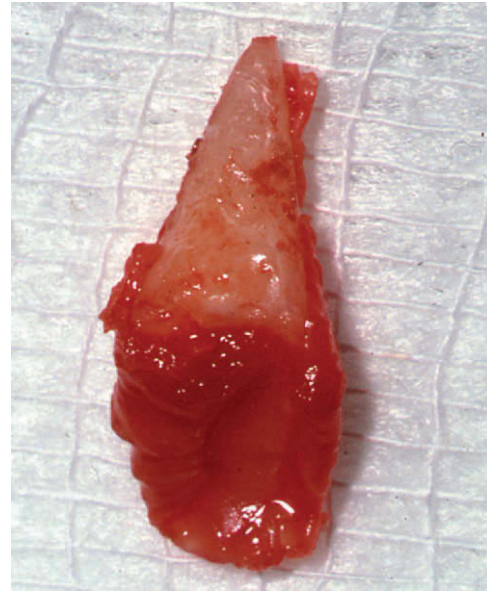
Although this was found to be successful in 9 of 12 cases that were treated by one investigator,<sup>5</sup> this method of treatment was suggested more than 30 years ago before the advent of single tooth endosseous implants, which may be considered a more predictable alternative today.

### TREATING THE CORONAL AND APICAL FRAGMENTS AT THE SAME TIME

It may be difficult to get a good apical seal when endodontically treating just the coronal fragment of a fractured tooth.



E



F



G



H

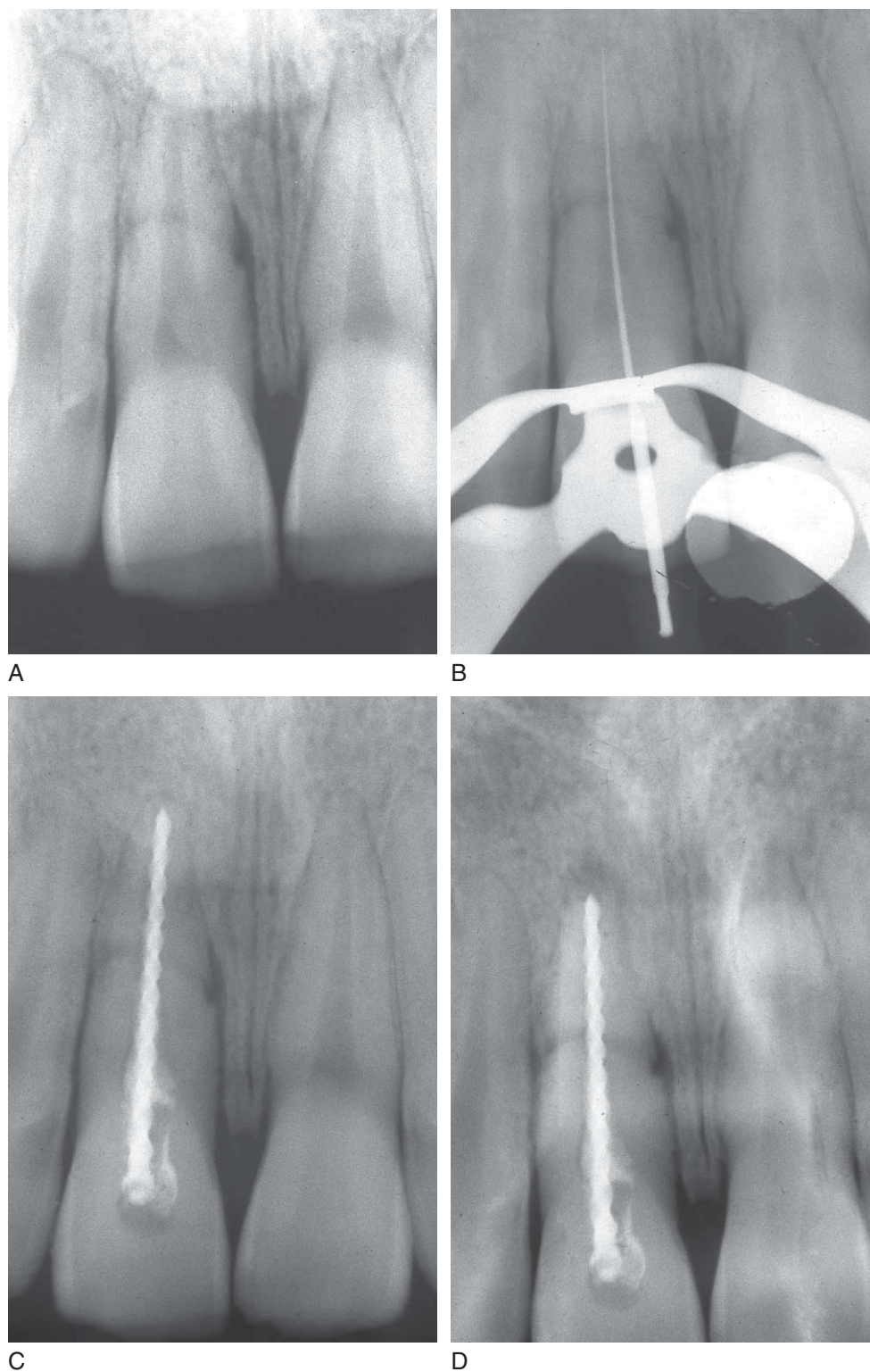
**Figure 4-12, cont'd** The apical fragment was surgically removed and the proximal granulation tissue was curetted (**E-G**). An external resorption defect was also repaired on the adjacent lateral incisor. **H**, The patient was asymptomatic 6 months later, with good bone deposition.

Trying to achieve this seal while simultaneously accessing and sealing the coronal *and* apical openings of the *apical* fragment is almost impossible. That is probably why this type of treatment has such a low success rate. There is little documentation in the literature of these types of cases, but

in a recent study involving only seven teeth treated in this manner, there was a zero percent rate of healing.<sup>20</sup>

It has been suggested that if the coronal and apical fragments are in a favorable approximation, an *intraradicular splint* should be used<sup>5,30</sup> (Fig. 4-13). This would involve canal





**Figure 4-13** An attempt to connect the coronal and apical fragments with an intraradicular splint (**A-C**). **D**, Six months later bone loss developed in the area of the fracture site and apically, indicating nonhealing of the endodontic treatment.

cleaning and shaping of both the coronal and apical fragments, and the placement of a stabilizing splint through both canal spaces. Similar to the difficulty in obtaining an adequate seal at the fracture site when simultaneously obturating both the coronal and apical fragments, an intraradicular splint also has this same challenge, and successful cases treated in this manner are anecdotal at best. In a study done more than 30 years ago, an investigator found success in five of six teeth treated with an intraradicular splint.<sup>5</sup> But the sample size was small and long-term follow-up of these cases would be necessary.

As previously described, some cases of intraalveolar root fractures heal by a calcific fusion of the coronal and apical fragments. Sometimes the pulp tissue within *both* of these fragments subsequently becomes nonvital, and treating the apical fragment can become difficult because of the lack of accessibility through the coronal fragment. Figure 4-14 shows a case in which the coronal fragment was conventionally treated, followed by endodontic treatment of the apical fragment through a *surgical* approach.

## REMOVING THE CORONAL FRAGMENT AND TREATING THE APICAL FRAGMENT

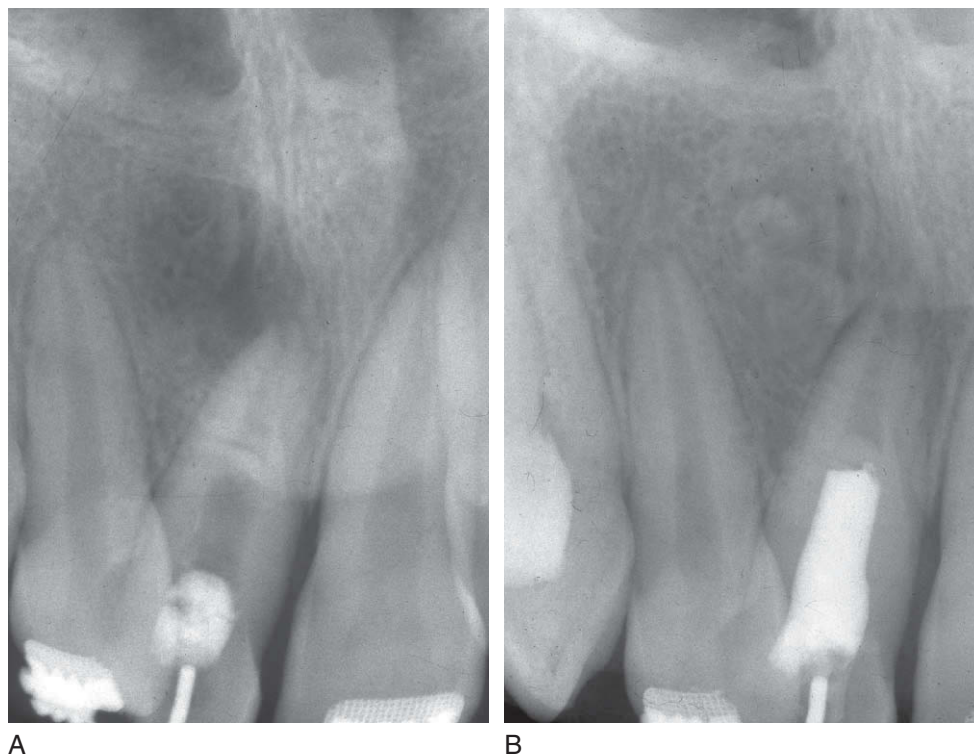
### Crown-Root Fractures

Commonly there are cases in which the traumatic injury, by direct or indirect impact, results in the crown of the tooth

becoming completely fractured off from the root at the gingival level or slightly below, and may extend into the area just below the crestal bone. These types of injuries have been called *crown-root fractures*<sup>2</sup> (Fig. 4-15). By definition, these cases are really *not* considered *intraalveolar root fractures* because the fracture is not completely encased within bone. However, often the treatment plan for these teeth is similar to that of an intraalveolar root fracture that occurs in the coronal third of the root. With injuries such as these, the coronal fragment may stay attached only by gingival fibers and will exhibit severe mobility. This fragment must be removed, usually under local anesthetic, and an assessment needs to be made as to the restorability of the remaining apical fragment. Assuming the apical fragment is restorable and will result in an acceptable crown-to-root ratio after a crown is fabricated (at least 1:1), then the optimal treatment plan would be to perform endodontic treatment on the remaining apical fragment and subsequently place a crown. As described below, other procedures, such as orthodontic extrusion and/or periodontal crown lengthening, may be necessary to facilitate the proper placement of a crown.

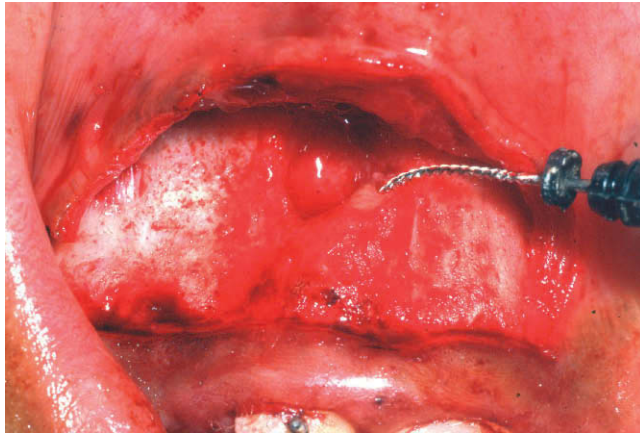
### Coronal Root Fractures

When there is an intraalveolar root fracture in the coronal third of the tooth that results in excessive mobility of the coronal fragment, sometimes the only option is to remove the coronal fragment. In many of these situations, the apical

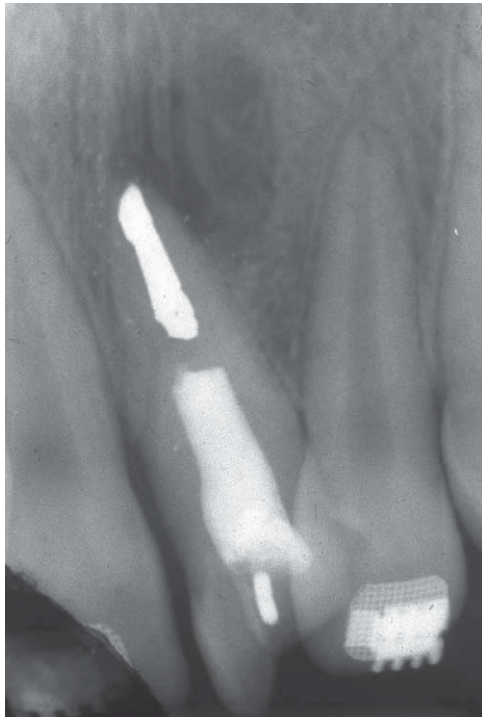


**Figure 4-14 A-D,** This patient presented with an access in the coronal fragment, and a calcific fusion of the coronal and apical fragments. The coronal fragment was treated conventionally, but there was subsequent pain and swelling apically. Because the calcific fusion prevented conventional endodontic treatment of the apical fragment, the apical fragment was treated surgically.

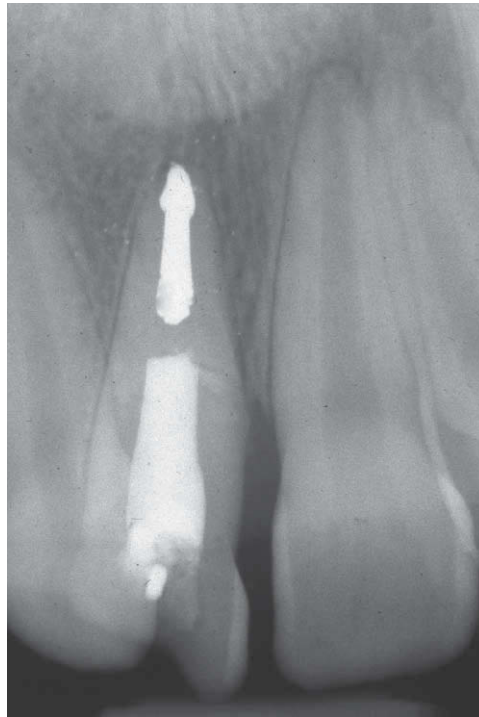
*Continued*



C



D



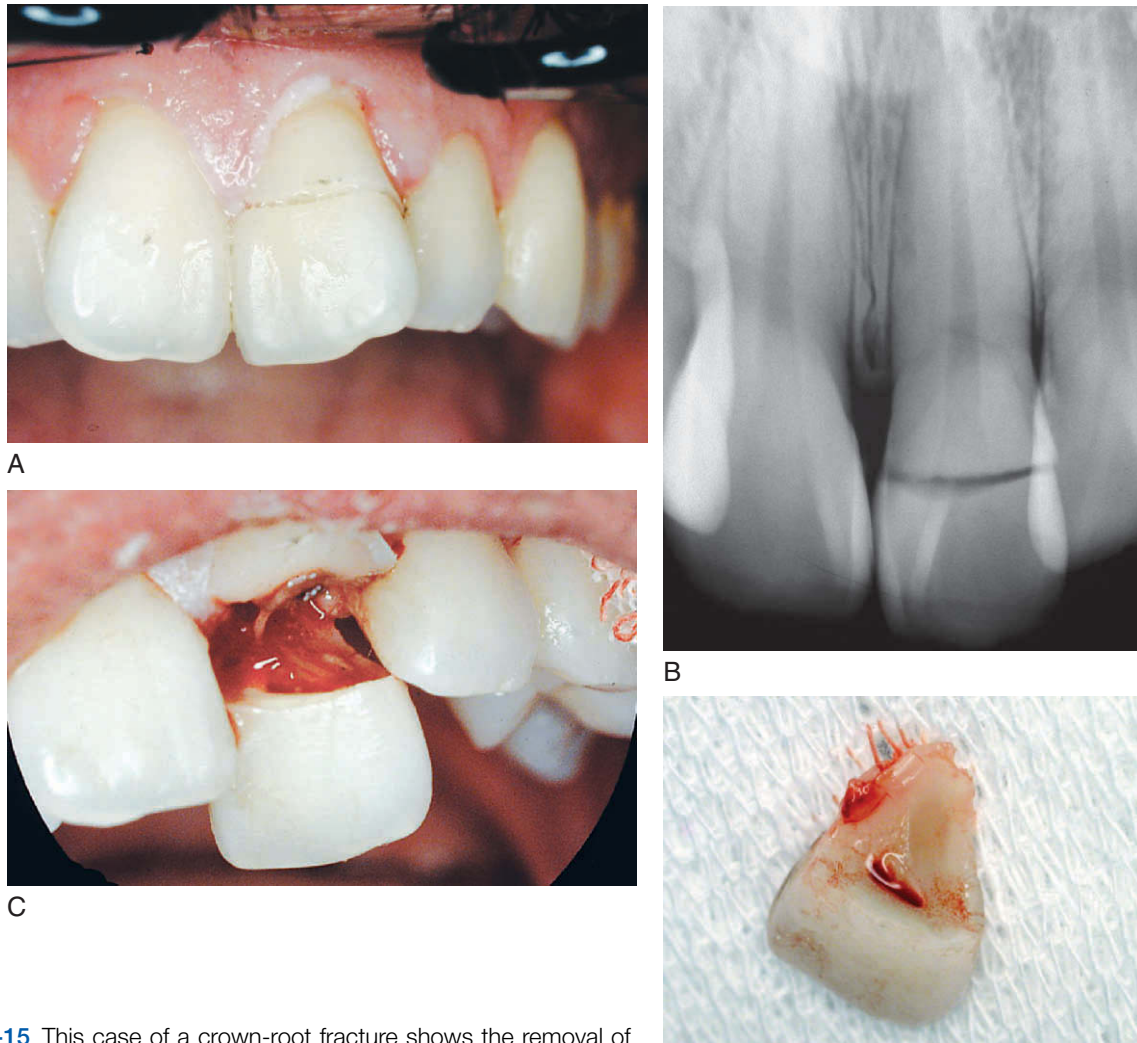
E

**Figure 4-14, cont'd E,** One year post treatment radiographs show good healing.

fragment may be both restoratively and endodontically salvageable. For these cases, to obtain an adequate crown margin on the apical fragment, periodontal crown lengthening may be necessary. This is typically accomplished by electrosurgery, laser surgery, or incisional periodontal surgery, often with osseous recontouring being necessary. Unfortunately, this may result in compromised esthetics from uneven periodontal contouring compared with the adjacent teeth. Extended clinical crowns may be an unfavorable cosmetic result, and the interdental papillae and interproximal bone may be compromised. One alternative to crown lengthening is to orthodontically extrude the apical fragment before restoring it<sup>22</sup> (Fig. 4-16). Orthodontic extrusion of a root, also known as *forced eruption*, is a relatively

simple form of orthodontic movement. Various orthodontic appliances have been recommended, both fixed and removable,<sup>24,36,47</sup> while maintaining adequate anchorage of the adjacent teeth. Generally the fixed appliances tend to be more predictable in their outcome.<sup>11,24</sup> The technique involves first performing endodontics on the apical fragment and the placement of a post. There is an attachment of various sorts on the end of the post that attaches to the extrusion appliance. Root movement usually extends over a 4 to 8 week period, generating about 1 mm of movement every 1 to 2 weeks, using orthodontic forces of 15 to 30 g.<sup>11</sup> When the orthodontic movement is slow, the alveolar bone and gingival attachment tends to move along with the root. This will often require some soft and hard tissue





**Figure 4-15** This case of a crown-root fracture shows the removal of the clinical crown that was fractured slightly below the crestal bone.

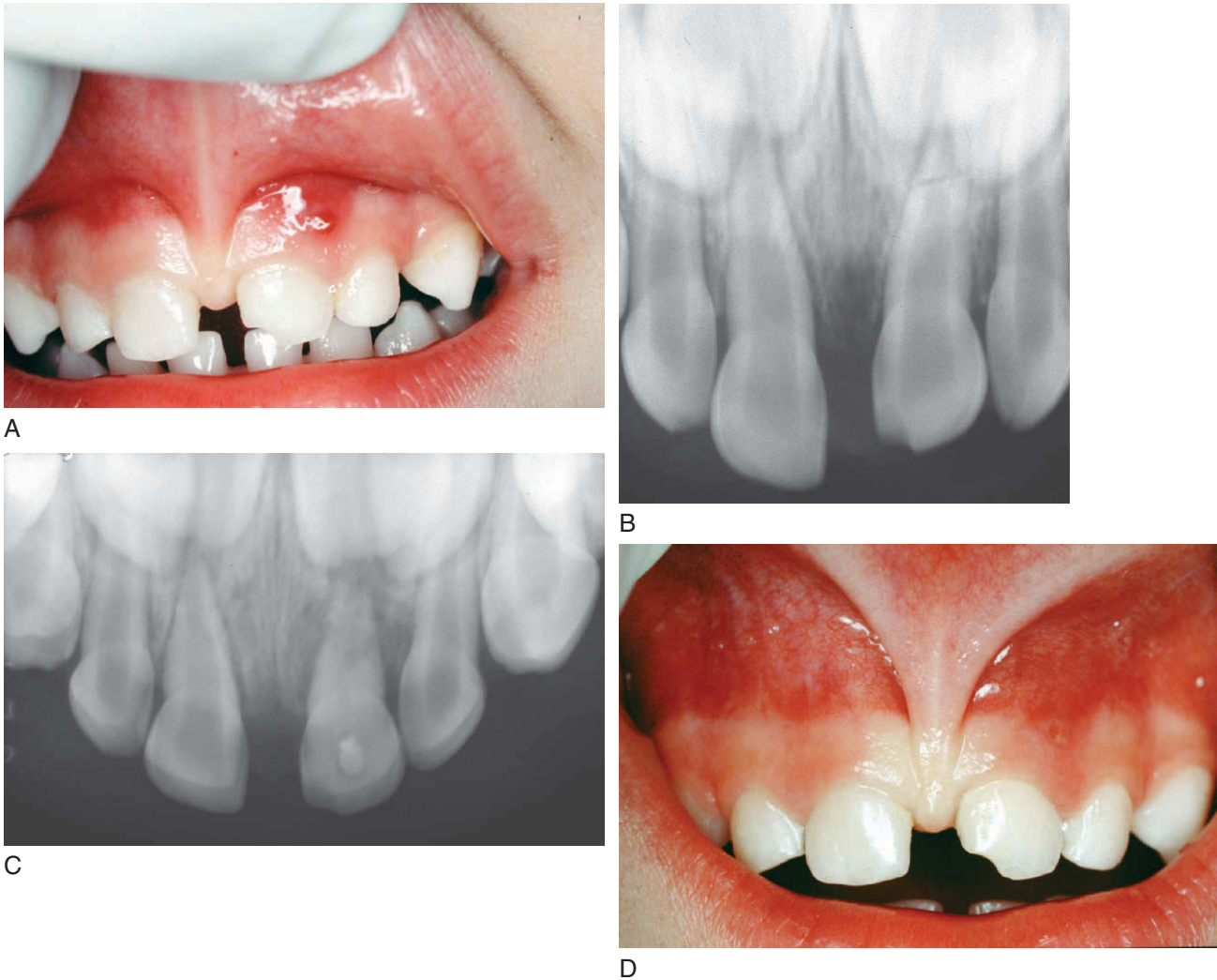
recontouring to achieve an acceptable crown margin, an acceptable biological width, and a favorable esthetic appearance of the gingival margin compared with that of the adjacent teeth. Once the extrusion is complete, a stabilization period is preferred to limit the relapsing intrusive tendency. It has been recommended that this stabilization period be 1 month for each millimeter of root movement.<sup>24</sup> There have been some suggestions in the literature that doing a supracrestal fiberotomy before, during, and after the forced eruption may decrease this intrusive tendency and decrease the time necessary for postmovement stabilization. However, the necessity and predictability of this hypothesis remains unproven.<sup>11,24</sup>

## PEDIATRIC CONSIDERATIONS

It is unusual to see horizontal intraalveolar root fractures in primary teeth. Typically, these teeth are luxated rather than fractured. However, in the event that this type of fracture does occur, care must be taken not to perform any procedure that may interfere with the proper development of the underlying succedaneous tooth. As with permanent teeth, these primary teeth tend to remain vital and asymptomatic, and typically no treatment is necessary. As the permanent tooth erupts, the fractured root of the primary tooth will resorb normally.<sup>5</sup> If treatment of the coronal fragment is considered, the canal space should be obturated with a mate-



**Figure 4-16** In the event of an intraalveolar root fracture when the coronal fragment is nonsalvageable, but there is adequate apical fragment remaining, orthodontic root extrusion may be the treatment of choice. **A-D** show the progress of extruding the apical fragment over a period of 6 months. **E** shows the restored apical fragment.



**Figure 4-17** A and B, Sinus tract associated with a nonvital primary central incisor that has an intraalveolar root fracture. C shows the endodontic treatment of the coronal fragment, with calcium hydroxide in place. D, The resolution of the sinus tract 3 months later.

rial that will absorb while the primary tooth resorbs and the underlying permanent tooth erupts (Fig. 4-17).

## CONCLUSION

Most teeth with intraalveolar root fractures do not require any endodontic treatment. There is a higher rate of healing when the fracture is more apical and when the fragments are more closely approximated. Healing most often involves a calcific joining of the fragments or the development of an intact lamina dura around the fragments with bone forming between the fragments. Splinting, if necessary, should only be semirigid and should not exceed 4 weeks. When endodontic treatment is required, it should involve only the coronal fragment with an emphasis on proper length control and a good apical seal from the obturation.

In advance of any treatment, the patient should always be informed as to the suspected prognosis, and given treatment options when available.

## REFERENCES

1. American Association of Endodontists: Glossary of endodontic terms, ed 7, 2003.
2. American Association of Endodontists: Recommended guidelines of the American Association of Endodontists for the treatment of traumatic dental injuries, American Association of Endodontists, October 2004.
3. Andreasen FM: Transient root resorption after dental trauma: the clinician's dilemma, *J Esthet Restor Dent* 15(2):80-92, 2003.
4. Andreasen FM, Yu Z, Thomsen BL: The relationship between pulpal dimensions and the development of pulpal necrosis



- after luxation injuries in the permanent dentition, *Endod Dent Traumatol* 2:90-98, 1986.
5. Andreasen JO: Treatment of fractured and avulsed teeth, *J Dent Children* Jan-Feb:29-48, 1971.
6. Andreasen JO, Andreasen FM, Mejare I et al: Healing of 400 intra-alveolar root fractures. 1. Effect of pre-injury and injury factors such as sex, age, state of root development, fracture type, location of fracture and severity of dislocation, *Dent Traumatol* 20:192-202, 2004.
7. Andreasen JO, Andreasen FM, Mejare I et al: Healing of 400 intra-alveolar root fractures. 2. Effect of treatment factors such as treatment delay, repositioning, splinting type and period and antibiotics, *Dent Traumatol* 20:203-211, 2004.
8. Andreasen JO, Hjorting-Hansen E: Intra-alveolar root fractures: radiographic and histologic study of 50 cases, *J Oral Surg* 25:414-426, 1967.
9. Arakawa S, Cobb CM, Rapley JW et al: Treatment of root fracture by CO<sub>2</sub> and Nd:YAG lasers: an in vitro study, *J Endod* 22:662-667, 1996.
10. Austin LT: A review of forty cases of retained fractured roots of anterior teeth, *J Am Dent Assoc* 17(4):1930-1932, 1930.
11. Bach N, Baylard J, Voyer R: Orthodontic extrusion: periodontal considerations and applications, *J Can Dent Assoc* 70(11):775-780, 2004.
12. Barclay C: Root resorption. 2: Internal root resorption, *Dent Update* 20(7):292-294, 1993.
13. Bender IB, Freedland JB: Clinical considerations in the diagnosis and treatment of intra-alveolar root fractures, *J Am Dent Assoc* 107:595-600, 1983.
14. Berman LH, Fleishman SB: Evaluation of the accuracy of the Neosono-D electronic apex locator, *J Endod* 10(4):164-167, 1984.
15. Bhaskar S, Rappaport H: Dental vitality tests and pulp status, *J Am Dent Assoc* 86:409, 1973.
16. Birch R, Rock WP: The incidence of complications following root fracture in permanent anterior teeth, *Br Dent J* 160:119-121, 1986.
17. Caliskan MK, Pehlivan Y: Prognosis of root-fractured permanent incisors, *Endod Dent Traumatol* 12(3):129-136, 1996.
18. Caliskan MK, Turkun M: Root canal treatment of a root-fractured incisor tooth with internal resorption: a case report, *Int Endod* 29(6):393-397, 1996.
19. Cvek M, Mejare I, Andreasen JO: Healing and prognosis of teeth with intra-alveolar fractures involving the cervical part of the root, *Dent Traumatol* 18:57-63, 2002.
20. Cvek M, Mejare I, Andreasen JO: Conservative endodontic treatment of teeth fractured in the middle or apical part of the root, *Dent Traumatol* 20:261-269, 2004.
21. Dederich DN: CO<sub>2</sub> laser fusion of a vertical root fracture, *J Am Dent Assoc* 130(8):1195-1209, 1999.
22. Delivanes P, Delivanes H, Kuftinec M: Endodontic-orthodontic management of fractured anterior teeth, *J Am Dent Assoc* 97:483-485, 1978.
23. Diangelis AJ, Bakland LK: Traumatic dental injuries: current treatment concepts, *J Am Dent Assoc* 129:1401-1414, 1998.
24. Durnam T, Goddard T, Marrison S: Rapid forced eruption: a case report and review of forced eruption techniques, *Gen Dent* March-April:167-175, 2004.
25. Feely L, Mackie IC, Macfarlane T: An investigation of root-fractured permanent incisor teeth in children, *Dent Traumatol* 19(1):52-54, 2003.
26. Fulling HJ, Andreason JO: Influence of maturation status and tooth type of permanent teeth upon electrometric and thermal pulp testing, *Scand J Dent Res* 84(5):286-290, 1976.
27. Fuss Z, Tsesis I, Lin S: Root resorption—diagnosis, classification and treatment choices based on stimulation factors, *Dent Traumatol* 19(4):175-182, 2003.
28. Herweijer JA, Torabinejad M, Bakland L: Healing of horizontal root fractures, *J Endod* 18(3):118-122, 1992.
29. Hovland EJ: Horizontal root fractures. Treatment and repair, *Dent Clin North Am* 36(2):509-525, 1992.
30. Howden GF: Intra-radicular splinting of fractured roots, *Br Dent J* 128(2):81-84, 1970.
31. Jacobsen I: Root fractures in permanent anterior teeth with incomplete root formation, *Scand J Dent Res* 84:210-217, 1976.
32. Jacobsen I, Kerekes K: Diagnosis and treatment of pulp necrosis in permanent anterior teeth with root fractures, *Scand J Dent Res* 88(5):370-376, 1980.
33. Kawai K, Masaka N: Vertical root fracture treated by bonding fragments and rotational replantation, *Dent Traumatol* 18(1):42-45, 2002.
34. Lindahl B: Transverse intra-alveolar root fractures: roentgen diagnosis and prognosis, *Odont Rev* 9:10-24, 1958.
35. Lundberg M, Cvek M: A light microscopy study of pulps from traumatized permanent incisors with reduced pulpal lumen, *Acta Odontol Scand* 38(2):89-94, 1980.
36. Mandel R, Binzer W, Withers J: Forced eruption in restoring severely fractured teeth using a removable orthodontic appliance, *J Prosth Dent* 47:269-274, 1982.
37. Marshall FJ: Root fracture: report of a case, *Oral Surg* 13:1485-1487, 1960.
38. Maroto M, Barberia E, Planells P et al: Treatment of a non-vital immature incisor with mineral trioxide aggregate (MTA), *Dent Traumatol* 19(3):165-169, 2003.
39. Michanowicz A, Michanowicz J, Abou-Rass M: Cementogenic repair of root fractures, *J Am Dent Assoc* 82:569-579, 1971.
40. Qin M, Ge L, Bai R: Use of a removable splint in the treatment of subluxated, luxated and root fractured anterior permanent teeth in children, *Dent Traumatol* 18(2):81-85, 2002.
41. Rafter M: Apexification: a review, *Dent Traumatol* 21(1):1-8, 2005.
42. Rintaro T, Kiyotaka M, Minoru K: Conservative treatment for root fracture located very close to gingiva, *Dent Traumatol* 21(2):111-114, 2005.
43. Robertson A, Andreasen FM, Bergenholtz G et al: Incidence of pulp necrosis subsequent to pulp obliteration from trauma of permanent incisors, *J Endod* 22(10):557-580, 1996.
44. Rock WP, Gordon PH, Friend LA et al: The relationship between trauma and pulp death in incisor teeth, *Br Dent J* 136(6):236-239, 1974.
45. Schwartz RS: Mineral trioxide aggregate: a new material for endodontics, *J Am Dent Assoc* 130(7):967-975, 1999.
46. Shabahang S, Torabinejad M, Boyne P et al: A comparative study of root-end induction using osteogenic protein-1, calcium hydroxide, and mineral trioxide aggregate in dogs, *J Endod* 24(1):1-5, 1999.
47. Simon J, Kelly W, Gordon D et al: Extrusion of endodontically treated teeth, *J Am Dent Assoc* 97:17-23, 1978.
48. Smith JW: Calcific metamorphosis: a treatment dilemma, *Oral Surg* 54(4):441-444, 1982.
49. Stalhane I, Hedegard B: Traumatized permanent teeth in children aged 7-15 years, *Swed Dent J* 68:157, 1975.
50. Steinig TH, Regan JD, Gutmann JL: The use and predictable placement of mineral trioxide aggregate in one-visit apexification cases, *Aust Endod J* 29(1):34-42, 2003.
51. Tegsjo U, Valerius-Olsson H, Olgart K: Intra-alveolar transplantation of teeth with cervical root fractures, *Swed Dent J* 2(3):73-82, 1978.

52. Welbury R, Kinirons MJ, Day P et al: Outcomes for root-fractured permanent incisors: a retrospective study, *Pediatr Dent* 24(2):98-102, 2002.
53. World Health Organization: Application of the international classification of diseases to dentistry and stomatology, ed 3, Geneva, Switzerland, 1995, Office of Publishing, World Health Organization.
54. Yates JA: Root fractures in permanent teeth: a clinical review, *Int Endod J* 25(3):150-157, 1992.
55. Zachrisson BU, Jacobsen I: Long-term prognosis of 66 permanent anterior teeth with root fracture, *Scand J Dent Res* 83:345-354, 1975.

# LUXATION INJURIES



ASGEIR SIGURDSSON AND CECILIA BOURGUIGNON

## CHAPTER OUTLINE

### CLASSIFICATION

### ETIOLOGY AND EPIDEMIOLOGY

### DIAGNOSIS OF LUXATION INJURIES: RADIOGRAPHIC AND CLINICAL ASPECTS

Concussion

Subluxation

Lateral Luxation

Lateral Luxation Without Apical Displacement

Lateral Luxation With Apical Displacement

Extrusive Luxation

Intrusive Luxation

### TREATMENT OF LUXATION INJURIES

#### Emergency Treatment

General Treatment Considerations

Repositioning

Concussed and Subluxated Teeth

Laterally Luxated Teeth

*Without Apical Displacement*

*With Apical Displacement*

Repositioning Extruded Teeth

Repositioning Intruded Teeth

*Spontaneous Eruption*

*Surgical Crown Uncovering*

*Orthodontic Extrusion*

*Surgical Extrusion*

Splinting

Suturing Soft Tissue Lacerations

Additional Considerations, Prescriptions, and Instructions to the Patient

### Posttrauma Follow-up Evaluations and Treatment

Recall Frequency

Endodontic Evaluation and Treatment

Mature Teeth (Closed Apex)

Immature Teeth (Open Apex)

### SEQUELAE OF LUXATION INJURIES

Types

Pulpal

Periradicular

Diagnostic Means to Detect Sequelae on Follow-up

### EXPECTED HEALING OUTCOME OF LUXATION INJURIES

### CONCLUSION



The term *dental luxation* refers to a group of distinct clinical situations in which the common presentation is a severance or disruption between the tooth and its surrounding tissues secondary to trauma. This may occur with or without visible tooth displacement. The resulting damaged tissues are the cementum, periodontal ligament (PDL), and pulpal neurovascular supply. Proper posttrauma management and healing of these tissues is imperative, and the dentist must direct the clinical approach to ensure that the prognosis of the affected tooth is maximized.

The factors that determine the type and severity of the luxation injury seem to be related to the force and direction of the impact. Furthermore, the resilience of the alveolar bone, which decreases with age, probably plays a role.<sup>17,62</sup> Severely luxated and displaced teeth tend to develop more extensive sequelae (e.g., pulp necrosis, root resorption, canal calcification, and/or cessation of root development of immature teeth) than teeth that suffer only a minor luxation and displacement.<sup>9,24,47</sup>

## CLASSIFICATION

Traditionally, all luxation injuries, including avulsions, have been classified into six main categories. These classifications are to some degree based on the *World Health Organization (WHO) International Classification of Diseases*,<sup>13</sup> but have been modified and adapted by Jens and Frances Andreassen<sup>6</sup> to include some entities that were not in the original WHO list. Based on the previous classifications, and for the purpose of diagnosis refinement, we describe these main categories as follows:

- **Concussion (N 503.20)\*:** an injury to the tooth-supporting structures, without abnormal loosening or displacement of the tooth. The tooth is sometimes sensitive to percussion.
- **Subluxation (N 503.20):** an injury to the tooth-supporting structures with abnormal loosening, but without displacement of the tooth. The tooth is usually sensitive to percussion.
- **Lateral luxation (N 503.20):** the tooth is displaced labially, lingually, distally, or mesially, with or without an associated apical displacement.
- **Extrusive luxation (N 503.20):** the tooth is displaced in an incisal direction, with or without a concomitant lateral luxation.
- **Intrusive luxation (N 503.21):** the tooth is displaced in an apical direction, into the alveolar bone.
- **Avulsion (N 503.22):** the tooth is completely displaced out of the alveolar socket.

These luxation injuries are categorized in an increasing order of severity. The severity of the injury correlates with the healing potential and prognosis of the affected tooth, particularly if inadequate treatment is performed. In this

chapter, the diagnosis and treatment of the first five categories of luxations will be discussed. Avulsions will be discussed in Chapter 6.

## ETIOLOGY AND EPIDEMIOLOGY

In dental traumatology, luxations are the most commonly underreported type of injury.<sup>7</sup> This is possibly due to the patients' perception that these injuries do not necessarily require the assistance of a dentist. Indeed, in many situations, patients are unaware of the injury's potential severity because there may be no visible signs of a tooth-related problem. Often the tooth is in its normal position, although sometimes tender to percussion or mastication.

Data collected on reported luxation injuries (including avulsions) indicate that they occur between 22% and 61% of all dental injuries, with the most commonly involved tooth being the maxillary central incisor.<sup>11,14,39,48</sup> The probable reason why the incidence of these injuries varies among the studies is that the data were collected and recorded in different types of settings. For example, when the data were collected in dental offices, there seemed to be a trend towards a lower incidence of luxation injuries and a higher incidence of crown fractures. When the data were collected in a hospital emergency room environment, the above trend of incidences was reversed. Presumably, when patients assume that their trauma is severe, they tend to seek treatment in a hospital emergency room facility rather than in a dental office. Additionally, the pertinent literature indicates that injuries of minor severity, such as concussions and subluxations, have been reported to constitute between 30% and 77% of all luxation injuries.<sup>10,11,20</sup> Again, this incidence variation reinforces the notion that the manner and setting in which the data are collected plays an important role in determining the type of injury reported.

Depending on the type of dental injury, there can be severe damage to the tooth, pulp, and surrounding structures.<sup>1,2,3,7,11</sup> The damage can sometimes be visualized immediately, but in some cases the damage may be revealed only weeks, months, or even years later. Therefore a thorough dental evaluation of even the mildest of injuries is essential and should include an evaluation of the adjacent and opposing teeth and their surrounding tissues. This must be performed immediately after trauma (to diagnose the injuries *per se*) and at subsequent follow-up visits<sup>9</sup> (to diagnose the injury sequelae).

## DIAGNOSIS OF LUXATION INJURIES: RADIOGRAPHIC AND CLINICAL ASPECTS

As stated in Chapter 2, before any dental evaluation, the injured patient must be evaluated by the dentist for more

\*Denotes the closest WHO category registration number.

serious injuries, especially to the central nervous system (CNS). A thorough history of the injury should then be gathered before any kind of dental treatment is initiated. If the mechanism of injury suggests more trauma than dental involvement, immediate referral to a medical facility is indicated.

Radiographic evaluation is always necessary in cases of dental injuries. But the number of radiographs necessary will depend upon the clinical presentation and type of injury. In some cases, a single periapical radiograph may be sufficient (i.e., a situation of obvious concussion injury or minor crown fracture). However, more severe injuries may require the exposure of up to four radiographs, including not only periapical radiographs, but also occlusal, panoramic, and/or lateral skull radiographs.<sup>1,5</sup> The periapical radiographs should be exposed at different angulations. The clinician will want to rule out any fracture of the root and/or alveolar bone, and often only one or two radiographs, especially if exposed at the same angulation, are not sufficient.<sup>12</sup> Additionally, the stage of root formation should be observed because the stage of root maturation directly influences the potential for pulp survival<sup>3</sup> and consequently the treatment options. Even in cases in which no posttraumatic damage is envisioned, initial periapical radiographs should always be taken for the purpose of future comparisons in the event that a pathosis develops.

## CONCUSSION

If a concussion has occurred, by definition the tooth is in its normal position and does not show any abnormal mobility. However, it may be tender to percussion or mastication. There should not be any bleeding around the sulcular area. These teeth present radiographically with a normal periodontal ligament width around the root.

Although the reported injury may have been described as “mild” by the patient, the perceived severity of the impact may not be reliable. The clinician should still examine the patient carefully and take the necessary periapical radiographs. Even if it seems that only one radiograph might be sufficient, it is prudent to expose a few additional radiographs from different angulations to rule out the possibility of a root fracture.

A recently concussed tooth may be unresponsive to pulp sensitivity tests, but this does not necessarily mean that the pulp is necrotic.<sup>46</sup> Pulpal sensitivity testing of a concussed tooth should always be performed, so that a base line for observing a pulpal recovery or a developing pathosis can be established.

## SUBLUXATION

Similar to a concussion injury, the subluxated tooth remains in its original position after the traumatic impact. However, some mobility is present and there might be some sensitivity to palpation on the facial aspect of the alveolus. The per-

cussion sound should be normal, although the tooth is very likely to be tender to percussion and mastication. Often there is minor bleeding from the sulcus around the tooth. Periapical radiographs should show that the periodontal ligament width is normal or only slightly thickened.

Intraalveolar root fractures may present themselves in an identical clinical pattern as subluxations. Therefore a proper radiographic and clinical assessment is essential.

Subluxated teeth may be unresponsive to pulp sensitivity testing initially.<sup>32,33,53</sup> Nonetheless, as described previously, immediate posttrauma and follow-up pulp sensitivity testing is imperative.

## LATERAL LUXATION

When a typical lateral luxation occurs, the tooth is displaced in either a facial or a lingual/palatal direction (the Z-axis) solely, but the cervical area of the tooth remains aligned with the adjacent teeth because there is no vertical displacement. However, in many instances, a lateral luxation is accompanied by some component of vertical displacement as well (i.e., extrusion or intrusion of the affected tooth). Anterior teeth are more commonly displaced lingually than facially.<sup>6</sup> This is related to the direction of the impact at the time of injury.

Traditionally, all types of lateral luxation injuries have been grouped together as if they were a single entity. This has led to some confusion with regard to determining the best treatment plan suited for each particular type of luxation. The issue of whether or not to perform endodontic treatment on a luxated tooth illustrates this confusion. The most important factor in determining the treatment plan for luxated teeth is *the presence or absence of significant apical displacement at the time of injury* (Figs. 5-1 and 5-2).

### Lateral Luxation Without Apical Displacement

These teeth are pushed only in a facial or lingual direction, with the apical root remaining in its original location within the socket. They are often loose enough to allow the clinician to move the tooth back to its original position with light digital force. Typically, there is some sulcular bleeding. Palpation will reveal normal contours of the alveolus. Although percussion testing may be unfeasible because of increased tooth mobility, if performed, it will usually generate a normal or slightly dull sound. When the luxated tooth's apex has remained in its original position and there is no widening of the PDL space (as confirmed by observing three periapical radiographs exposed from different angulations along with one occlusal radiograph), then there is a good likelihood that the neurovascular bundle is intact. Consequently, endodontic therapy will probably be unnecessary.

The pulp of laterally luxated teeth without apical displacement may be unresponsive to cold or electrical sensitivity tests initially and during the following weeks or sometimes months.<sup>33,36,53</sup>



A



B

**Figure 5-1** A and B, Two radiographs of a luxated incisor, exposed at different angulations, reveals that its apex is in its original position in both radiographs.

### Lateral Luxation With Apical Displacement

In these situations, the tooth is frequently pushed palatally or lingually and is firmly located in its new position. When percussed, it will elicit a dull metallic sound, audibly different compared with the adjacent teeth. This occurs because the apex has broken into the facial cortical plate and wedged the tooth in there. Palpating the alveolar bone plates may

indicate the new location of the apex. Proper radiographic evaluation, especially using occlusal radiographs, is imperative to ascertain the presence of apical displacement. Periapical radiographic examination will most likely show a PDL space that is widened around the midportion and coronal portion of the root, but normal or mildly compressed apically. If there is radiographic evidence that the apex has moved out of its normal position, then there is a very high probability that the neurovascular bundle has been compromised. Consequently the tooth is likely to require root canal therapy, irrespective of whether it is mature or immature. However, immature teeth should be given a chance to revascularize. Therefore posttrauma radiographic and clinical monitoring is essential.

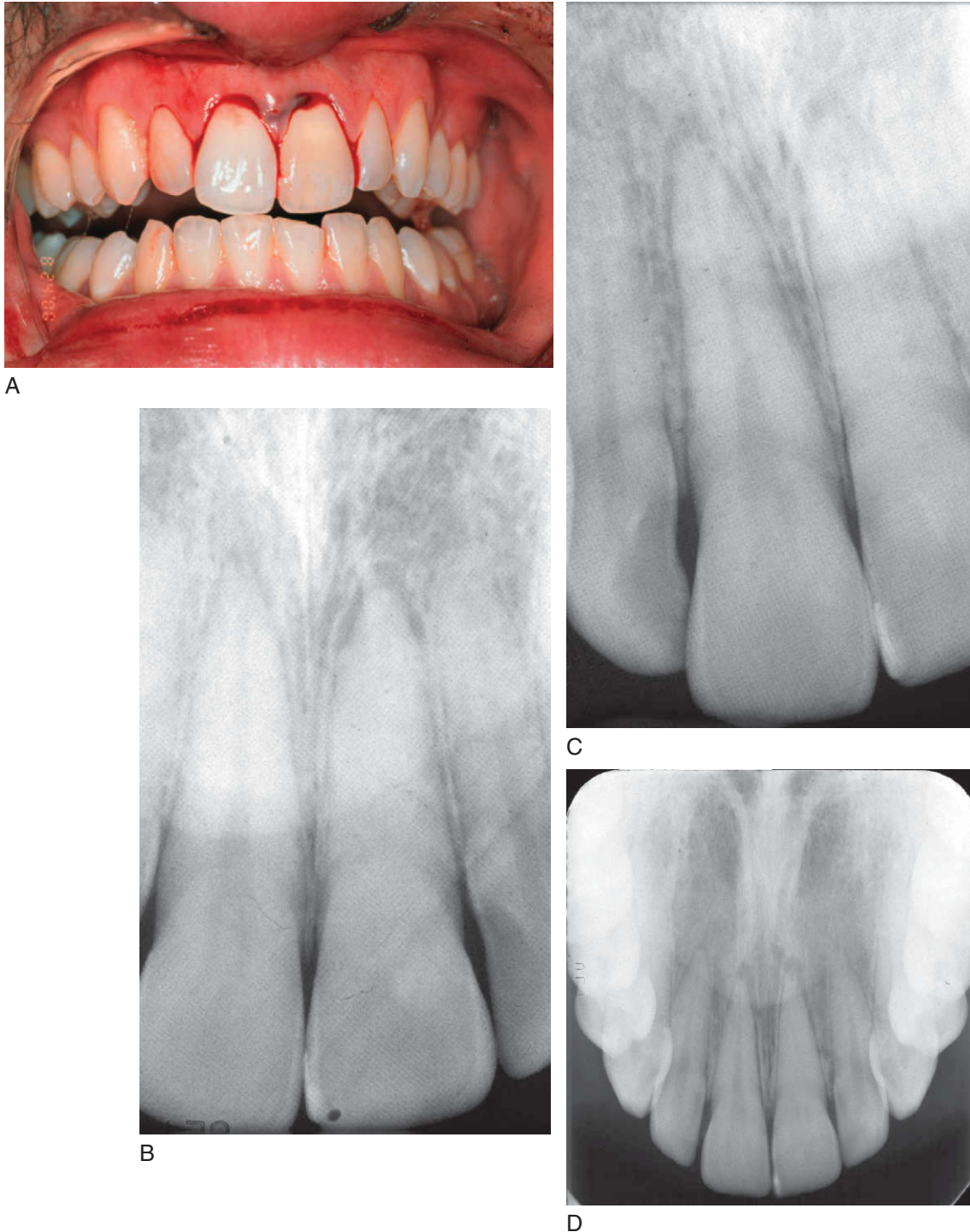
Laterally luxated teeth with apical displacement do not respond to cold or electric pulp testing initially and, because of the severity of the injury to the neurovascular bundle, will usually remain nonresponsive. Therefore in the case of a tooth with complete root formation (closed apex), the patient should be advised that endodontic treatment is likely to be necessary. In the case of an immature tooth (open apex), on some rare occasions pulpal sensitivity is regained after a few months or even after a year, if pulp revascularization occurs. These teeth should be monitored and endodontic treatment should be instituted immediately if signs of pulp necrosis and/or of root resorption are present.

### EXTRUSIVE LUXATION

In extrusions, the tooth will be loose and markedly extended out of its socket in comparison with adjacent teeth. Lateral luxations are frequently associated with extrusions. It has been stated that if a tooth moves more than 2 mm in an axial direction, there is little hope for pulp survival, particularly if the involved tooth displays a fully formed apex.<sup>19</sup> Although there are no scientific data to support this, it appears that the further the tooth moves vertically, the less likely the pulp vascularity will remain intact.

A thorough clinical and radiographic examination is very important because it may be questionable whether the *entire* tooth is extruded or whether there is an extruded coronal fragment of an intraalveolar root fracture (see Chapter 4). Periapical radiographs will most likely show a widened periodontal ligament space on the mesial and/or distal aspects of the root, and there may be an “empty” radiolucent space apically (Fig. 5-3). Depending on how far the tooth has been extruded, the pulp may or may not respond to pulp tests. The greater the extent of the extrusive luxation, the greater the likelihood that the tooth’s neurovascular bundle has been severed.<sup>11</sup> Therefore before any treatment, it is important to document how much the tooth has moved away from its original position. The clinician should also ask if the tooth has been repositioned back to its socket before the dental visit.





**Figure 5-2** **A**, Luxated maxillary left central incisor. Note sulcular bleeding. **B** to **D**, Three different radiographic angulations of the same luxated tooth, showing that the apex has moved out of its original position.

## INTRUSIVE LUXATION

An intruded tooth will appear as if it is not fully erupted, such that a portion or even the whole crown is submerged subgingivally. The tooth is firmly locked into the alveolar bone. A thorough radiographic evaluation of intruded teeth

is necessary and will typically show complete disappearance of the PDL space. As a result of the massive injury to the neurovascular bundle, intruded teeth will not respond to pulp sensitivity tests.<sup>6</sup>

From a diagnostic point of view, it can sometimes be difficult to determine whether the clinical presentation



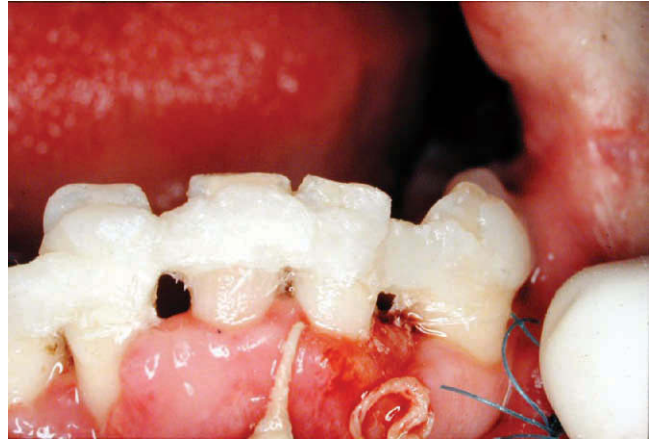
A



B



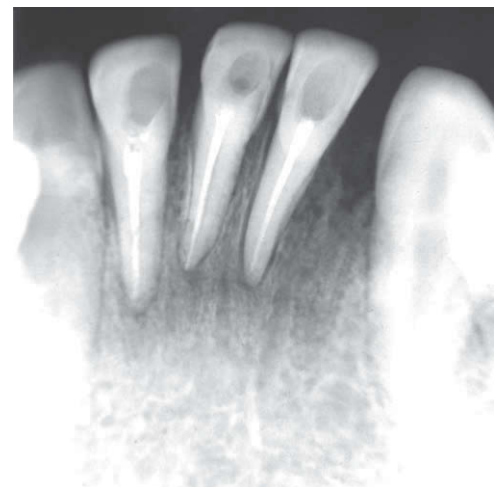
C



D



E



F

**Figure 5-3** Extrusive luxation of teeth #s 24, 25, and 26, and avulsion of #23. **A**, One week posttrauma, with inappropriate wire splint. **B**, One week posttrauma after splint removed. **C**, Radiograph after splint removed. Note apical sockets from which teeth were extruded. **D**, New splint application. **E**, Splint removal after 1 month. **F**, Radiograph 8 months posttrauma after endodontic treatment. Note regeneration of bone apically and in area of avulsed tooth.



A



B



C

**Figure 5-4** Intrusive luxation. **A**, Extensive edema of the upper lip secondary to alveolar bone compression and fracture. **B**, Patient profile shows loss of definition of lip and base of nose secondary to edema. **C**, Vestibular hematoma secondary to alveolar fracture and gingival laceration.

corresponds to a traumatic intrusion or to an incompletely erupted tooth. Intrusive luxations typically display the following clinical characteristics (Fig. 5-4):

- Edema of the lip from alveolar process fracture, decreasing the delineation of the upper lip and the base of the nose (for maxillary anterior teeth).
- Hematoma of the vestibule and lip secondary to the fracture of the alveolar process.
- Laceration of the associated gingiva.

This is particularly true when examining children and adolescents. The percussion test is useful in these circumstances because intruded teeth will elicit a distinctive metallic sound when percussed compared with adjacent teeth. A completely intruded tooth may also be mistaken for an avulsed tooth or a subgingival crown-root fracture. In some cases, the root apex may even be pushed through the nasal floor, causing bleeding from the nasal cavity.

The degree of intrusion should be carefully recorded in two ways:

1. Measure how far the intruded crown extends apically from the gingival margin.
2. Measure the distance from the intruded tooth's new location to the incisal edge of adjacent teeth.

Every time the patient is reevaluated, these measurements should be repeated and recorded. If the tooth does not spontaneously move towards its original position over a period of 3 to 5 weeks, then immediate treatment is indicated as described below.<sup>18</sup>

When primary teeth are intruded, the clinician should try to determine if their apices were forced facially or palatally. Often, they are pushed through the thin facial plate away from the forming permanent tooth. Occasionally it can be seen that the permanent tooth bud may be severely affected by an intrusive luxation, requiring immediate treatment (Fig. 5-5). A lateral jaw radiograph is useful for the purpose of confirming whether the intruded primary tooth has been pushed facially or towards the underlying permanent tooth bud. However, this may not be feasible with young children. Indeed, in a recent study of very young children, it was concluded that lateral extraoral radiographs should not be used routinely in intrusion cases of primary incisors because they were not diagnostically helpful for two main reasons: (1) the evaluation was difficult as a result of overlapping when multiple teeth were intruded and/or when the intruded teeth were lateral incisors, and (2) clinical examination and periapical radiographs were sufficient to allow an accurate





A



B



C

**Figure 5-5 A to C,** Permanent teeth with a history of intrusion of their primary predecessors into the forming permanent tooth buds, showing enamel changes and discoloration. (Courtesy Dr. William Vann.)

diagnosis in almost all cases.<sup>28</sup> The investigators advised that the clinician should make the diagnosis based on clinical findings and on examination of a periapical radiograph.

Of all luxation injuries, intrusion is the most devastating. This is because the PDL is severely crushed as the tooth is forced into the conically shaped socket and because in almost all circumstances the neurovascular bundle will be severed.<sup>5</sup> The more intrusive the luxation, the poorer the prognosis.<sup>30</sup>

## TREATMENT OF LUXATION INJURIES

### EMERGENCY TREATMENT

#### General Treatment Considerations

The main objective of the emergency treatment of luxation injuries is to reposition the tooth using minimal additional

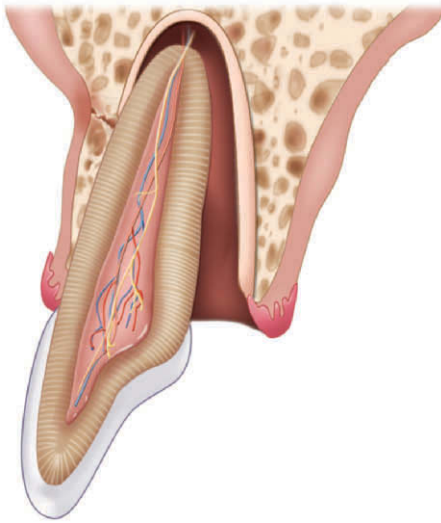
trauma to the PDL. Pulpal considerations are not a major issue during this emergency phase, although proper repositioning and adequate overall emergency care favor pulpal survival and healing.<sup>9</sup>

Once the patient has been evaluated, local anesthesia should be administered. It is advisable not to use a vasoconstrictor-containing anesthetic because it has been shown, for example, that 1:100,000 epinephrine-containing local anesthetics will diminish or completely stop the blood circulation in a healthy anterior pulp for up to 40 minutes.<sup>41</sup> Any additional tissue stress provoked by a compromised blood flow is undesirable for the injured pulp and surrounding tissues.

### Repositioning

#### Concussed and subluxated teeth

Concussed teeth should always be examined, but they do not require emergency treatment.



**Figure 5-6** Laterally luxated tooth with its apex still in its original position.

Subluxated teeth usually do not require emergency treatment either, except in unusual situations in which mastication or biting is painful. In these cases, a slight reduction of the occlusal interferences is indicated. With very young patients or patients who are mentally compromised, the clinician may find it necessary to splint the subluxated tooth for a few days to prevent the patient from further injuring it and its PDL.

### Laterally luxated teeth

**Without apical displacement** The tooth should be gradually moved back to its original position, using local anesthetic as necessary (Figs. 5-6 and 5-7). If there is any resistance, the associated alveolar bone socket should be carefully evaluated. If a socket fracture is present, the clinician should gently manipulate the tooth, pushing it and the alveolar bone back to normal position. Often, it is necessary to place a thin spatula into the space between the tooth and the socket to reposition the cortical plate to make room for the root. The tooth is then splinted in its original position, as described later in this chapter.

**With apical displacement** In these situations, the tooth is nearly always firmly lodged in its new location and it is almost impossible to push it directly forward to its original position (Figs. 5-8 and 5-9). The tooth presents with a high pitched metallic sound when percussed. The explanation for this is that the apex has broken through the cortical bone plate and stays wedged in there (Fig. 5-8, A). Concomitantly a major portion of the cervical and midroot PDL fibers remain attached to the root and socket, thus creating further

traction towards the apical area and thereby wedging the tooth even further.

It is very important to get the luxated tooth back to its original position as soon as possible, although this may be difficult when apical displacement is present (see Fig. 5-8, B). There may be a lot of bleeding in the socket where the apical root used to be; once the blood clots and organizes itself, in a matter of few hours, it may preclude the proper repositioning of the tooth. Although the tooth may be orthodontically moved back to its normal position at a later time, it might be an impractical solution. Furthermore, it may be unpleasant for the patient to maintain the tooth in a maloccluded position during that interim. To reposition the tooth back to its original location, the apex needs to be released from the cortical plate by pulling or pushing it slightly in a coronal direction. If the apex protrudes extensively through the bone cortical plate, it might be possible to push it in and downwards using the thumb, while concomitantly applying a gentle pressure to the palatal side of the crown with the index finger. Once the apex pushes away from the cortical plate, the tooth may become loose and in many cases it will snap back to its original position (see Fig. 5-8, C and D). Alternatively, if it is not possible to digitally manipulate the tooth back into its normal position, an extraction forceps may be useful in enabling the repositioning (see Fig. 5-8, E). Only a slight downward pull is recommended. Any rotational motion should be avoided, and no attempt should be made to move the tooth into place until it has become loose. If the tooth snaps back to its normal position, usually no splinting is required. Slight mobility as such will not compromise the tooth or its healing potential. The only reason for splinting these cases would be for patient comfort.

### Repositioning extruded teeth

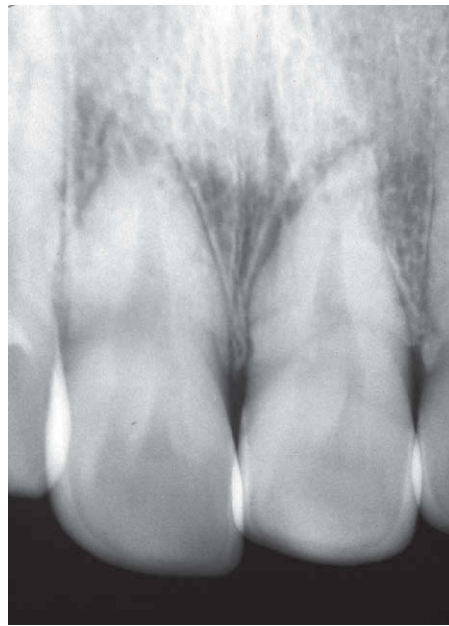
The extruded tooth should be gently repositioned into the socket. If a few hours have elapsed since the traumatic injury, there may be some apical resistance to repositioning the tooth. This is due to bleeding and the formation of a blood clot within the socket. The tooth should be gradually manipulated into the socket with digital pressure. Holding it securely in place with a formed pink wax plate, while the splint is being attached, is an easy way to counter the tendency of the tooth to drift out of the socket (Fig. 5-10). This technique is described later in this chapter under *Splinting*.

### Repositioning intruded teeth

Intrusions are the most severe of all luxations injuries so far discussed. They require that treatment decisions be made quickly and accurately.



A



B



C



E



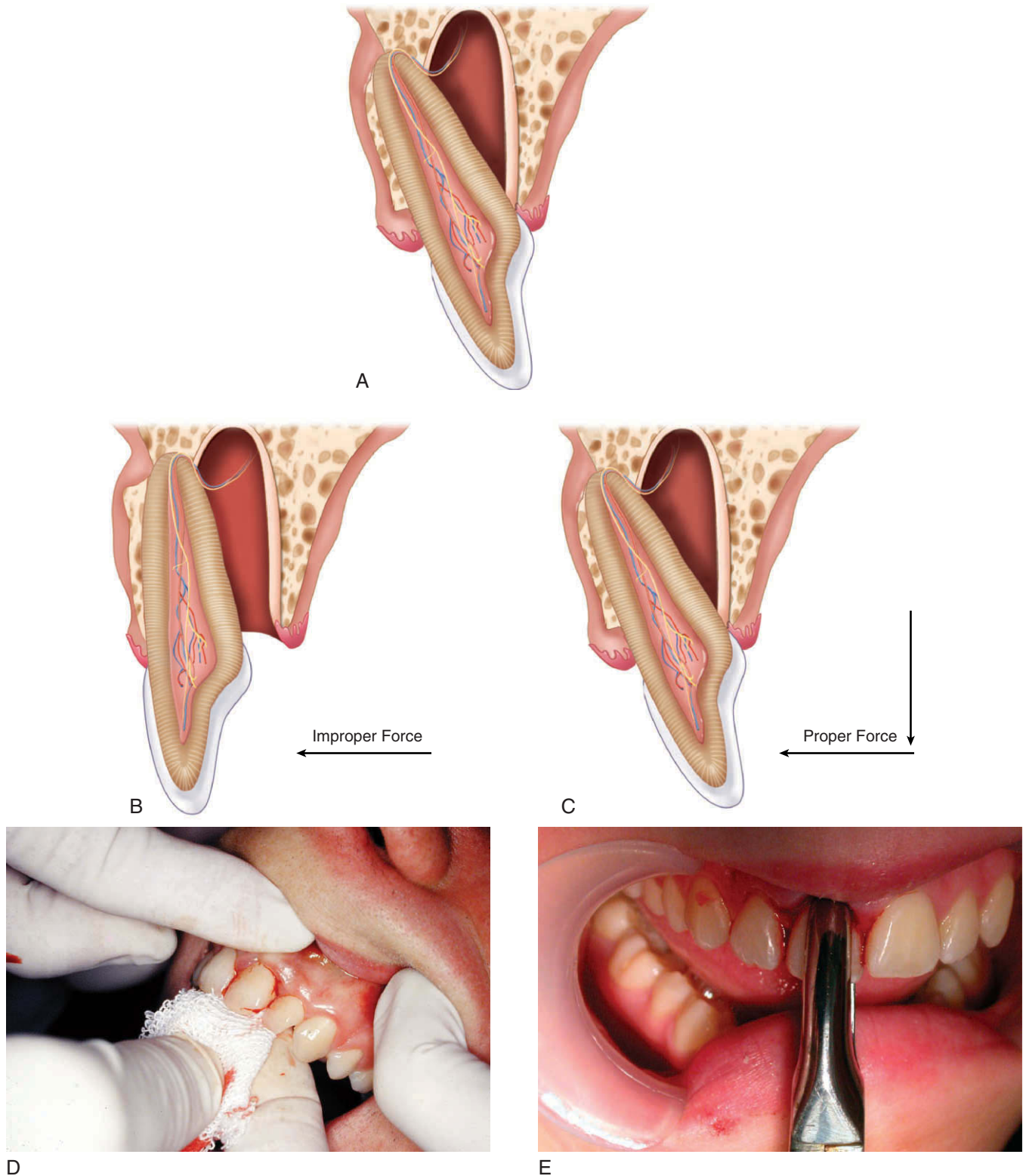
D



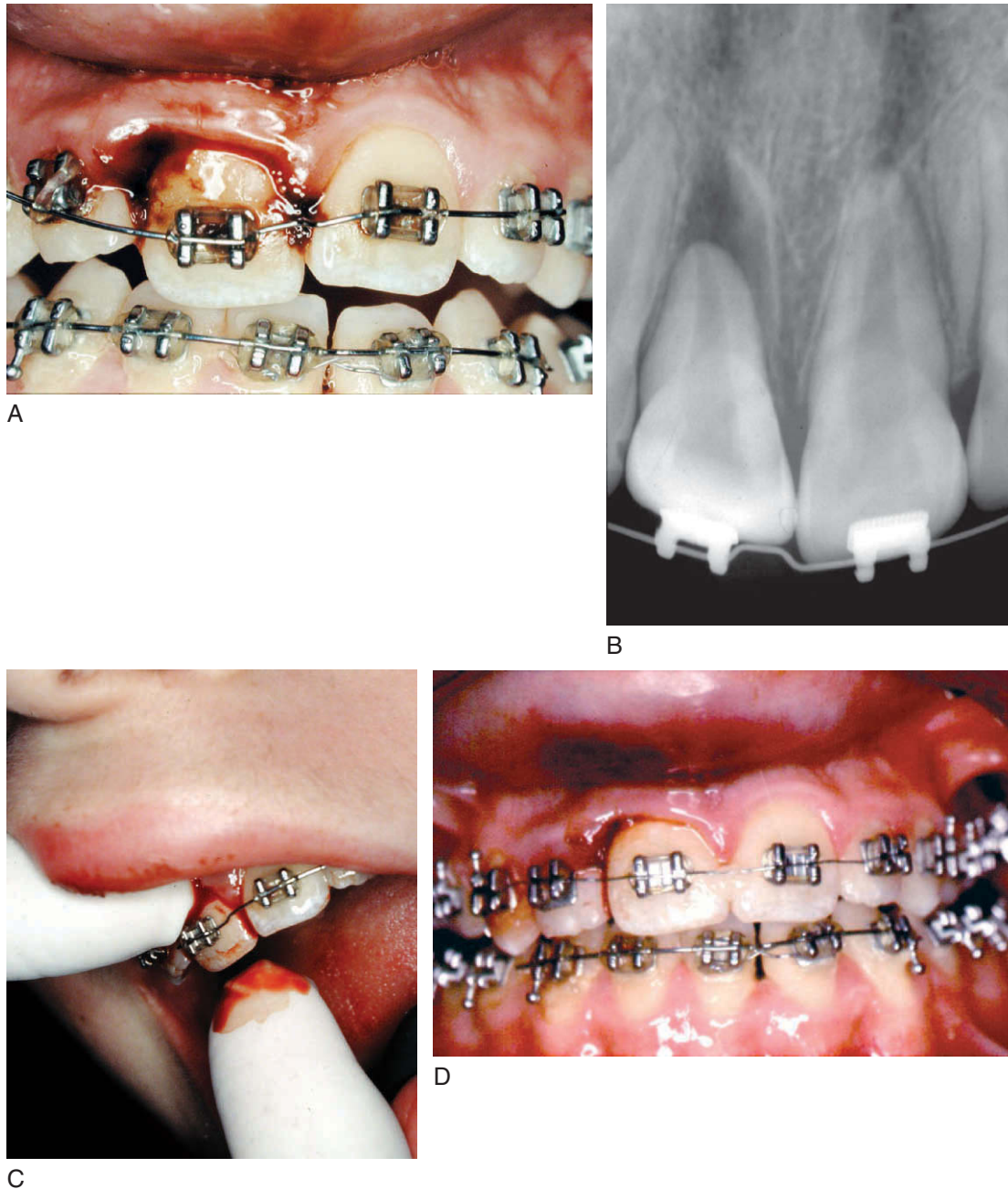
F

**Figure 5-7** Lateral luxation without apical displacement of maxillary left central incisor. **A**, Note occlusal interference from palatal luxation. **B**, Radiograph showing minimal apical displacement. **C**, Manual reduction of luxated tooth by applying palatal pressure. **D**, Radiograph showing tooth in proper position. **E**, Splint fabrication using Fiber-Splint (Polydentia S.A., Switzerland) and composite. **F**, Two-year follow-up radiograph showing good healing following endodontic treatment.





**Figure 5-8** Laterally luxated tooth with apical displacement. **A**, The apex broke through the cortical bone plate. **B**, Repositioning the tooth by pushing it straight forward will not allow it to return to its original place. **C**, The apical hold in the bone needs to be released first. This can be done either by pushing the apex coronally or by pulling the crown with an extraction forceps. Once the apex is freed, it is possible to rotate it with a forward motion. The tooth is then likely to swing back to its correct position and to firmly remain there because of the periodontal ligament fibers that have remained attached. **D**, If the apex of the tooth has penetrated through the cortical bone plate, it is necessary to push the apex down with the thumb finger, while at the same time pushing it gently forward with the index finger. **E**, If the tooth is firmly positioned in the cortical bone plate and if it is impossible to push it down coronally, then it must be pulled slightly with an extraction forceps. Once it becomes loose, it will easily go back to its original position. Care must be taken to pull the tooth straight downwards, with minimal or no rotation.



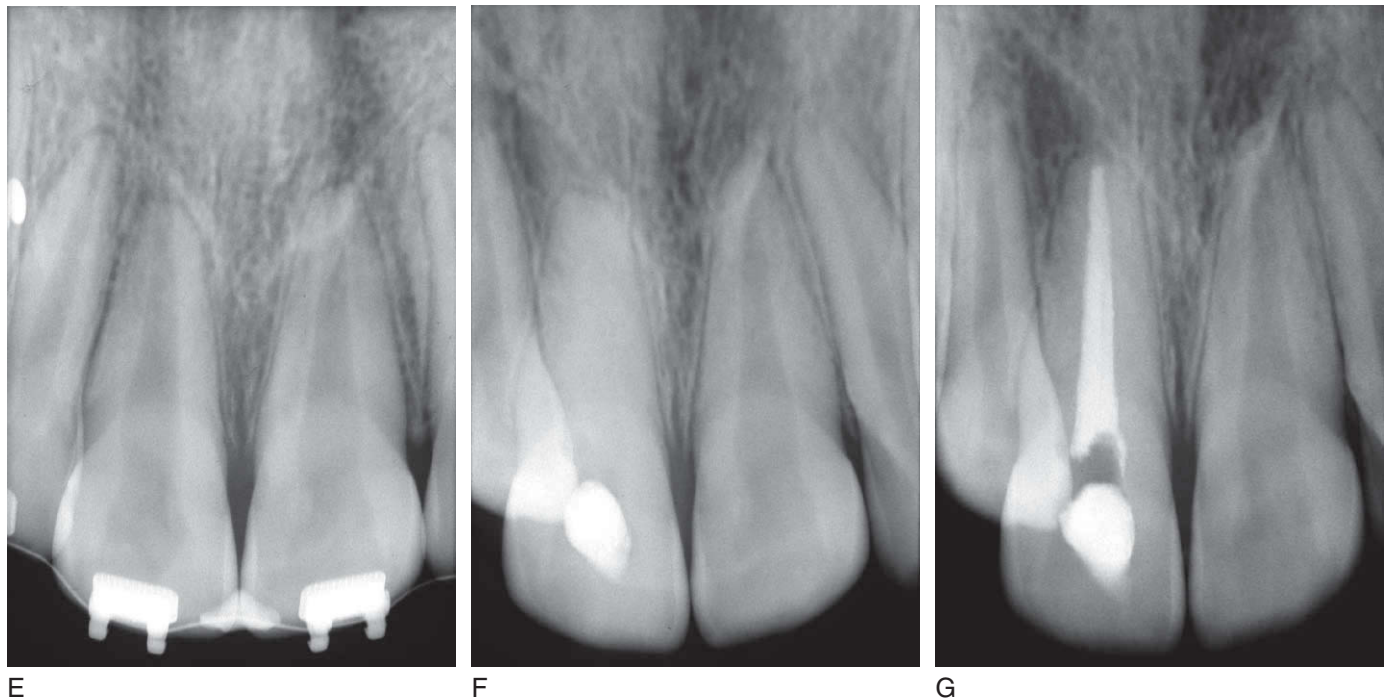
**Figure 5-9** Lateral luxation with apical displacement of maxillary right central incisor. **A**, Observe occlusal interference from luxation. **B**, Radiographic appearance of displaced apical root from alveolus. **C**, Manual reduction of luxated tooth. **D**, Tooth in proper position and splinted. Note vestibular hematoma from alveolar fracture. *Continued*

When managing an intruded *primary tooth*, the clinician must determine if the apex has been pushed through the facial cortical plate (Fig. 5-11). In these cases, there is a good chance that the tooth will reerupt spontaneously, and consequently no immediate treatment would be necessary.<sup>29</sup> However, if there are suggestions that the apex is directed towards or into the forming permanent tooth or tooth bud, then extraction of the primary tooth is the best option. There is a high likelihood that the permanent tooth bud has already sustained some damage during the injury

itself. But removing the primary tooth reduces the risk of further impeding the development of the succedaneous tooth, especially if the primary tooth becomes necrotic and infected.

When managing an intruded *permanent tooth*, there are basically four main treatment options:

- Allow for spontaneous eruption.
- Perform surgical crown uncovering.
- Perform orthodontic extrusion.
- Perform surgical extrusion.



**Figure 5-9, cont'd** **E**, Radiograph showing tooth in proper position. **F**, Radiograph showing endodontic treatment with calcium hydroxide applied within the canal. **G**, Radiograph showing final canal obturation 2 years after initial trauma. Note apical root resorption.



**Figure 5-10** Pink wax plate used to hold the extruded tooth in place while splinting.

**Spontaneous eruption** Even if a few case reports suggest the contrary,<sup>23,25</sup> there is growing evidence that the further the permanent tooth has been intruded, especially if it has a fully formed apex, the less likely it is to erupt on its own without complications.<sup>35</sup> The trouble with waiting for the tooth to reerupt spontaneously is that a pathologic fusion known as *ankylosis* or *replacement resorption* may develop between the

root surface and the bony socket (see Chapter 6). This fusion may start to occur in a matter of a few weeks. Once ankylosis has established itself, there is no chance for the tooth to erupt spontaneously, and the resulting prognosis is generally poor. If the tooth has been intruded less than 3 millimeters, there is some indication that it can survive and reerupt on its own<sup>45</sup> (Fig. 5-12). But if it is intruded more than 6 millimeters, spontaneous eruption without complications is unusual.<sup>35</sup>

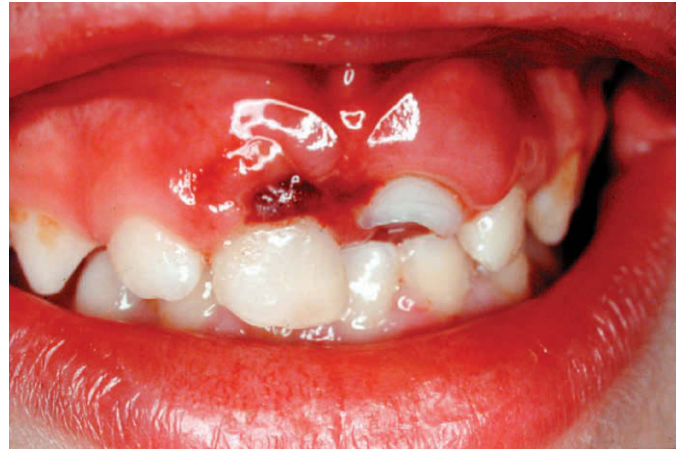
**Surgical crown uncovering** Surgically exposing the crown, immediately or shortly after an intrusive luxation, has been suggested to facilitate spontaneous reeruption, but there are no published studies to support this.<sup>25</sup> It is conceivable that this approach could be helpful for teeth that still have an open apex. Nonetheless, very careful posttraumatic monitoring is still mandatory and, if any signs of ankylosis develop, immediate action like orthodontic or surgical repositioning is imperative.

**Orthodontic extrusion** If orthodontic extrusion is planned for an intruded tooth, it should be initiated as soon as possible, delaying no more than 3 to 4 weeks posttrauma. There are only a few studies evaluating the true efficacy of this approach. One study<sup>61</sup> using a dog model indicated that severely intruded teeth showed signs of ankylosis after 11 to 13 days despite the initiation of orthodontic movement 5 to 7 days after the injury. However, less severely intruded teeth





A



B



C

**Figure 5-11** Intrusion of primary teeth. **A**, Profile of patient after intrusion, noting loss of definition between upper lip and base of nose from edema. **B**, Clinical photograph of intruded primary left central incisor. **C**, Radiograph revealing intrusion, uncomplicated crown fracture, and possible close proximity of root and developing permanent tooth bud.

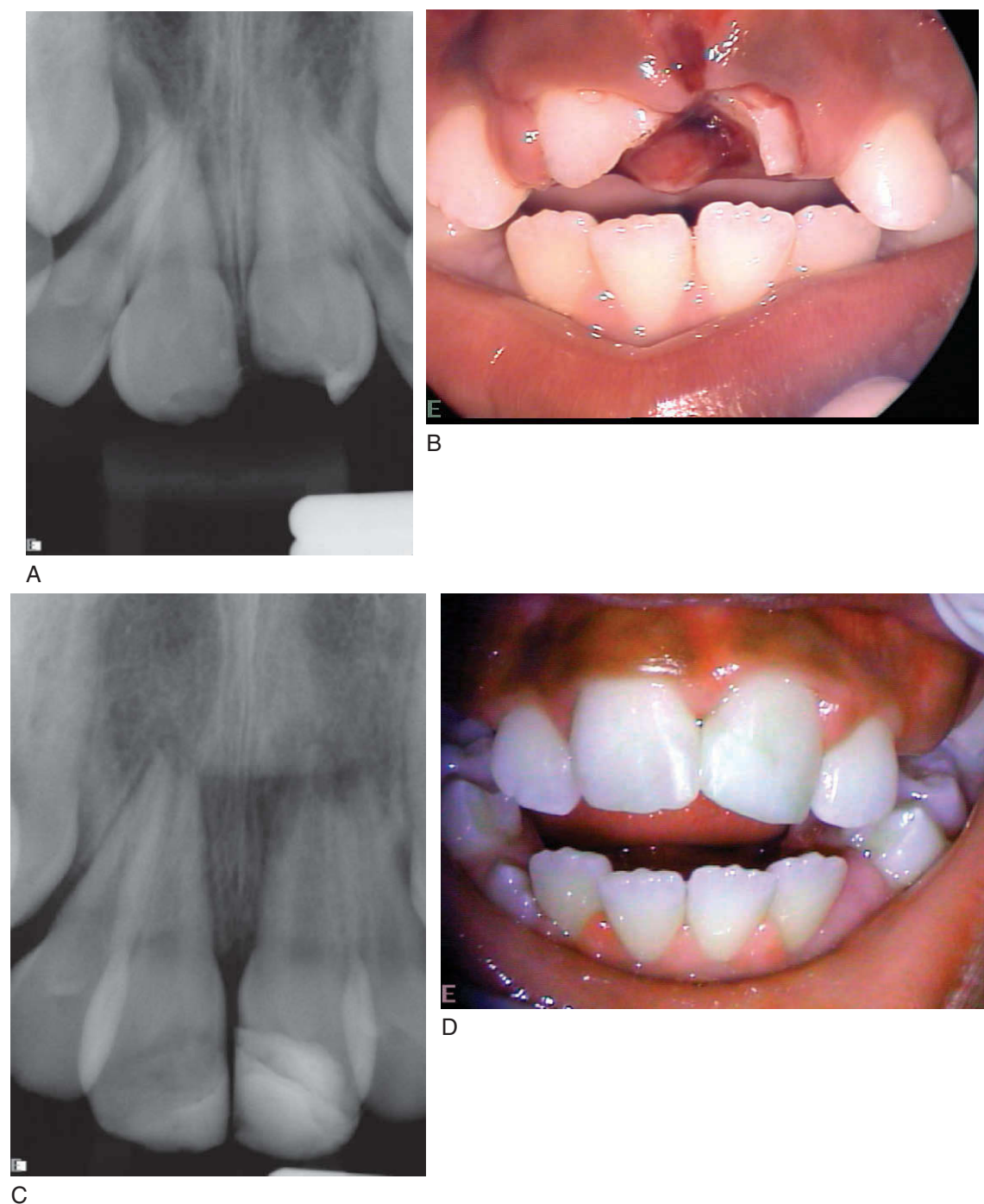
responded well to the orthodontic movement. Additionally, it has been suggested<sup>31</sup> that an intruded tooth should first be gently luxated before the placement of an orthodontic appliance. The tooth is then moved over a period of a few weeks to a maximum of 2 months, followed by a long stabilizing period of up to 2 or 3 months.<sup>18</sup> Meta-analysis of available case reports in the literature suggests that orthodontic extrusion can offer a predictable outcome,<sup>18</sup> but there is no published comprehensive study as of yet on the true efficacy of this approach.

**Surgical extrusion** In a recent study,<sup>21</sup> again using a dog model, it was stated that “a careful immediate surgical repositioning of a severely intruded permanent tooth with complete root formation has many advantages with few disadvantages.” Another investigation,<sup>23</sup> a retrospective study

of 58 intruded teeth, indicated that surgical repositioning resulted in a predictable outcome, with only 5 of the teeth being lost over the observation period. However, it was also observed that less surgical manipulation positively influenced healing (Fig. 5-13).

### Splinting

Splinting is generally not recommended for concussion or subluxation injuries.<sup>43</sup> Laterally displaced teeth without apical displacement, extruded teeth, and surgically repositioned intruded teeth require splinting. If applied, a splint should be flexible, should allow some tooth mobility, and should be removed within 7 to 10 days.<sup>6</sup> In cases of severe intrusion with surgical repositioning, and of luxation injuries associated with an alveolar bone fracture, a longer



**Figure 5-12** Intrusive luxation of 9-year old male with immature tooth development. **A**, Radiograph exposed 3 hours after intrusive luxation of teeth #8 and 9. Note uncomplicated crown fractures. **B**, Clinical photograph 3 hours after trauma. **C**, Radiograph 4 months after trauma with spontaneous re-eruption, but with apical root resorption of the left central insisor. **D**, Clinical photograph 4 months after trauma with teeth restored.

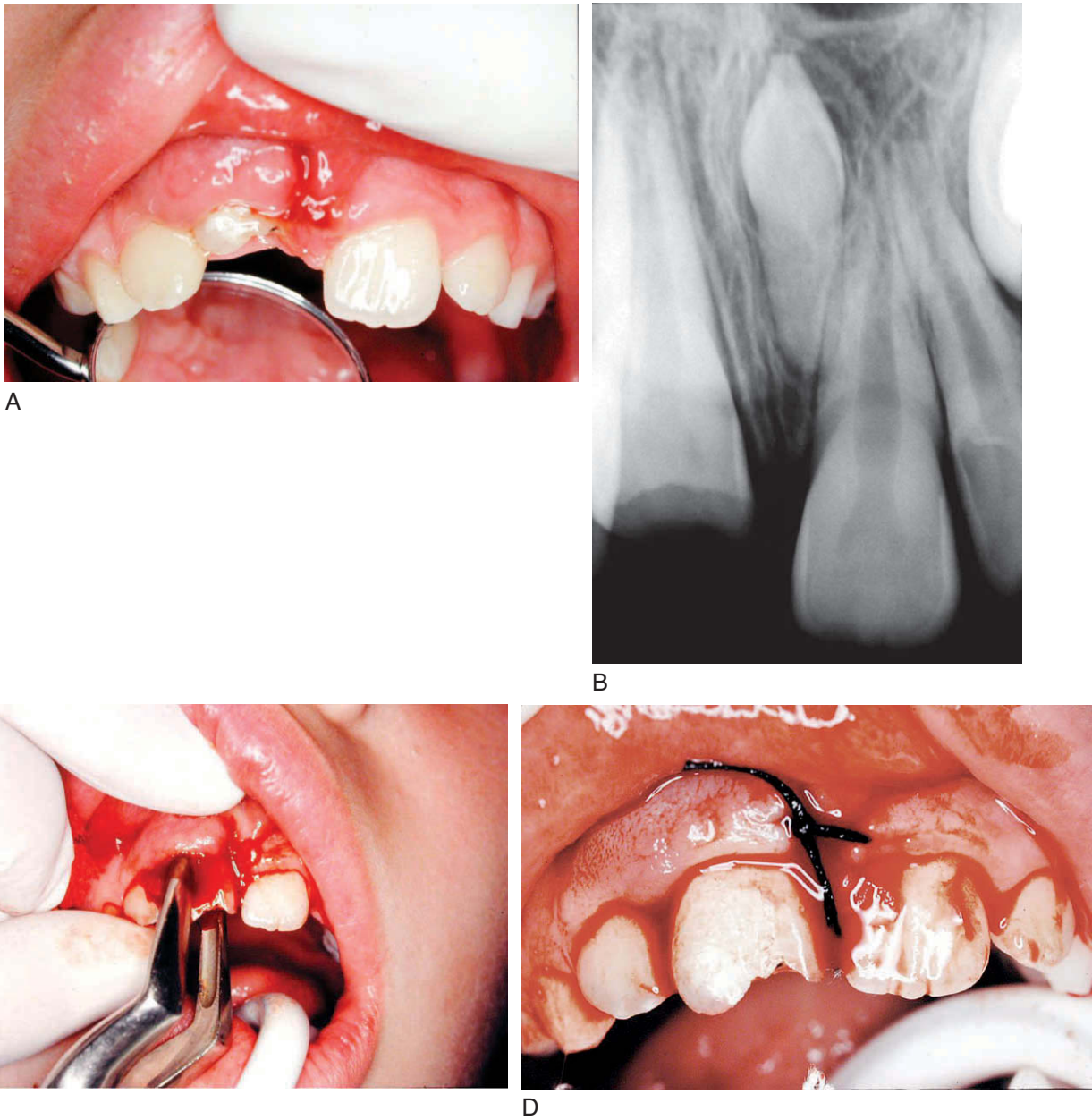
splinting time is advisable, but should not exceed a duration of 5 to 6 weeks.<sup>6</sup>

Many types of splints are available, but they must meet the following criteria:

- Easy to apply
- Easy to remove

- Nonimpinging on the gingiva
- Allow for good oral hygiene

The most commonly used splint consists of a soft wire or a 20- to 30-lb nylon fishing line, which is attached to the teeth with composite,<sup>6</sup> or direct application of composite between adjacent teeth (Fig. 5-14). Recently, more elaborate



**Figure 5-13** Maxillary right central incisor with intrusive luxation and surgical extrusion of tooth. **A**, Clinical appearance of intrusive luxation and uncomplicated crown fracture and gingival laceration. **B**, Radiograph showing intrusion, compressed PDL, and mesiodens. **C**, Repositioning of tooth with extraction forceps. **D**, Tooth in position with sutured gingival laceration. Today nylon sutures are recommended, not silk as shown here. *Continued*

splints have become available. One such splint uses Ribbond™ (Ribbond Inc., Seattle), a polyethylene fiber mesh, which is applied on the teeth and reinforced with composite (Fig. 5-15). This type of splint can be technique sensitive and also has the undesirable tendency to become rigid. Another splint system is called the *Titanium Trauma Splint*™ (TTS, Medartis AG, Basel, Switzerland). It consists of a titanium mesh that is designed to allow some movement of the teeth in the facio-lingual direction but not in the apical-incisal direction (Fig. 5-16). Its placement and

removal are easy, but this splinting system has the disadvantage of having a relatively high cost compared with the more traditional wire (or fishing line) and composite splint.

One of the main difficulties associated with splinting is keeping the teeth stable in their intended position while the splint is being placed. Splinting the tooth while having the patient bite into a warm pink wax plate during the procedure allows for a very stable platform to work on (Fig. 5-17). At the end of the emergency visit, after the splint has been placed, another periapical radiograph should be exposed to





E



F



G

**Figure 5-13, cont'd** E, Radiograph after tooth reduction and fixation, showing space between root and bone where bone had compression fractured secondary to traumatic intrusion. F, Radiograph 2 years after trauma and endodontic treatment. G, Restored tooth.



A



B

**Figure 5-14** A, Composite splint with a 30-lb fishing line. B, Direct attachment of a luxated tooth to adjacent teeth using composite bridge. Although easy to apply, this may be difficult to remove.



**Figure 5-15** A to E, Application of Ribbond™ fiber splint with composite. (Courtesy Drs. M. Miller and H. Juric.)

confirm the adequate repositioning of the tooth and to provide a basis of comparison for subsequent radiographic evaluations.

### Suturing Soft Tissue Lacerations

Lacerated gingiva and mucosa should be sutured with special attention given to the soft tissue closely surrounding the luxated tooth. This favors periodontal healing and prevents a portal of entry for bacteria to penetrate into the damaged tissue. The management of other soft tissue injuries is discussed in Chapter 8.

### Additional Considerations, Prescriptions, and Instructions to the Patient

Many luxated teeth, particularly displaced mature teeth, will require endodontic treatment. However, if indeed necessary, endodontic treatment should not be initiated at the emergency visit, but rather at a follow-up appointment 7 to 10 days later. As described later, this prevents further trauma to the tooth and its PDL during access and root canal preparation, prevents prolonging the treatment, and precludes the immediate placement of intracanal calcium hydroxide because this may have a detrimental effect on periodontal healing if applied in the canal space too early.



**Figure 5-16** Titanium trauma splint (TTS).

The pulp of immature luxated (displaced or nondisplaced) teeth may survive, heal, or revascularize after the injury. Consequently, as described later, no endodontic treatment of these teeth should be performed right away.

As stated previously, at the end of the emergency visit, once the splint has been installed, a reproducible periapical radiograph with minimal distortion should be taken. This is both to confirm the adequate repositioning of the luxated tooth and to establish a reference for subsequent evaluations.

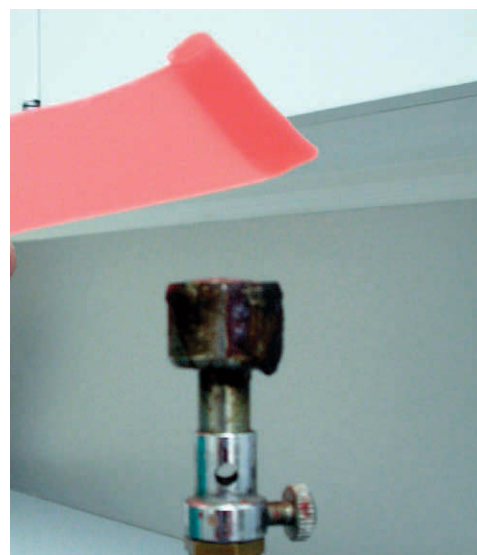
The patient should be prescribed appropriate analgesics, given instructions regarding oral hygiene, and be asked to rinse with 0.12% chlorhexidine twice a day for at least 1 week.<sup>59</sup> Although the use of antibiotics has not been recommended for luxation injuries, recent findings on avulsed teeth suggest that doxycycline can have a favorable effect on diminishing root resorption.<sup>52</sup> Since the PDL in a severe luxation is similar to that of an avulsion, doxycycline may have the same beneficial effect on luxated teeth as well.<sup>35</sup>

In addition to patient instructions following the emergency visit, the patient should be informed about the expected prognosis. Although predictions may be questionable, the patient has the right to know if the prognosis is thought to be poor, and may consider extraction as an alternative. This is discussed further in Chapter 9 under the concept of “patient informed consent.”

## POSTTRAUMA FOLLOW-UP EVALUATIONS AND TREATMENT

### Recall Frequency

All luxation injuries should be reevaluated after the injury, irrespective of the apparent severity of the injury. Table 5-1 shows the recommendations of the International Association of Dental Traumatology for a recall schedule after each type of luxation injury.



A



B

**Figure 5-17** Patient bites into a heated pink wax plate (A) to stabilize the luxated tooth while it is being splinted in its original position (B).

A meticulous clinical examination should be performed at each follow-up visit. It is important that pulpal vitality be assessed at each of these appointments because it has been shown<sup>49,53</sup> that the pulp can become nonvital weeks or months after an injury and also that the pulp can regain sensitivity a few weeks or months after trauma.

Radiographic evaluations are also necessary, so that the root surfaces can be observed for any signs of resorption or periapical/periradicular bone loss.

### Endodontic Evaluation and Treatment

Deciding the appropriate endodontic approach for the management of luxated teeth can be challenging for the clinician.

The type of luxation injury and the stage of root development will be the two key factors determining whether endodontic treatment will be more likely.



**TABLE 5-1** FOLLOW-UP SCHEDULE AFTER LUXATIONS

| TIME                   | UP TO 3 WEEKS | 3-4 WEEKS | 6-8 WEEKS | 6 MONTH | 1 YEAR | YEARLY FOR 5 YEARS |
|------------------------|---------------|-----------|-----------|---------|--------|--------------------|
| Concussion/subluxation |               |           | C         |         | C      |                    |
| Lateral luxation       | S+C           |           | C         | C       | C      | C                  |
| Extrusive luxation     | S+C           |           | C         | C       | C      | C                  |
| Intrusive luxation     | C             | C         | C         | C       | C      | C                  |

Adapted from International Association of Dental Traumatology: Follow-up procedures for traumatized permanent teeth. Available at: <http://www.iadt-dentaltrauma.org>.

S, Splint removal; C, clinical and radiographic examination.

In the case of a mild concussion or subluxation injury, and even in the case of a slight lateral or extrusive luxation, there is a good chance that the pulp will survive and maintain normal function, irrespective of the tooth being mature or immature.<sup>7,8</sup>

#### Mature teeth (closed apex)

In cases of lateral luxation with apical displacement, intrusive luxation, or severe extrusive luxation of a mature tooth, there is little chance for pulp survival.<sup>7</sup> Endodontic therapy should therefore be initiated between 7 and 10 days post-trauma to prevent the necrotic pulp from becoming infected.<sup>53</sup>

The 7- to 10-day delay in endodontic treatment is necessary because: (1) additional manipulation of the tooth could further traumatize the tooth and PDL, besides adding treatment time to the emergency visit, and (2) the early application of calcium hydroxide, the most commonly used intracanal medicament in dental trauma, can have a detrimental effect on PDL healing if applied too early.<sup>38</sup> Time must be allowed for the PDL to reattach.<sup>40</sup>

Because the luxated tooth may still be somewhat loose 7 to 10 days after the trauma, it is in most cases easier to keep the tooth splinted while it is being endodontically treated. The splint can then be removed at the end of the appointment, if so indicated.

If endodontic therapy is necessary, the tooth should be accessed aseptically, with rubber dam isolation, and instrumented to the appropriate size. It is not advisable to clamp the traumatized tooth. Currently the most widely accepted intracanal medicament is calcium hydroxide paste, either premixed or mixed with 0.12% chlorhexidine or sterile water.<sup>16,22,27,54,56</sup> It has been shown that if calcium hydroxide is placed into the root canal system of a traumatized tooth before becoming infected, and kept in place for 2 weeks to several months, the treatment outcome is favorable.<sup>60</sup> If an established infection exists before the placement of calcium hydroxide, then it is recommended that the calcium hydroxide remain in place for several months before the final obturation.<sup>59</sup>

#### Immature teeth (open apex)

Because of the larger vascular supply in immature teeth, pulpal survival or revascularization is a greater possibility subsequent to a luxation injury<sup>3,7,24,55</sup> (see Fig. 5-12). This would be a desirable mode of healing. However, waiting for a favorable outcome increases the risk of pulp canal infection and external inflammatory root resorption, which can severely compromise the tooth.<sup>7,11,55,57</sup> Frequent reevaluations are advised and should be performed in even shorter time intervals than those recommended in Table 5-1.<sup>26</sup> If signs or symptoms of pulp necrosis and infection appear, such as excessive mobility, sensitivity to palpation and percussion, periapical radiolucency, swelling or discomfort, or nondevelopment of the root, then endodontic treatment should be instituted as soon as possible.<sup>57</sup> This often involves an apexification procedure (see Chapters 3 and 6 for apexification treatment and for alternative endodontic approaches on necrotic pulps of immature teeth).

## SEQUELAE OF LUXATION INJURIES

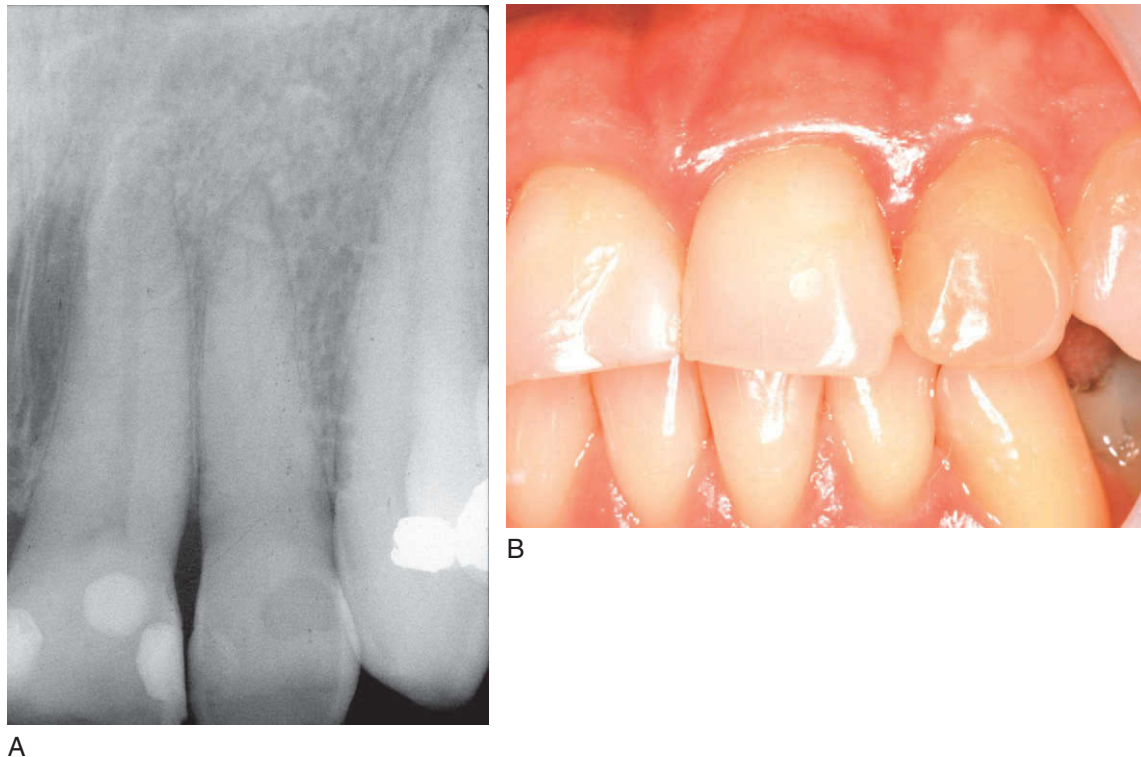
### TYPES

#### Pulpal

Potential pulpal sequelae following luxation injuries include pulp necrosis and pulp canal obliteration, each of which occurs in 35% to 40% of all luxated teeth.<sup>37,42</sup>

In an 11-year follow-up study of teeth that sustained luxation injuries, Andreassen<sup>3</sup> concluded that pulpal healing could be divided into 3 groups according to the degree of injury directed to the pulp: mild, moderate, or severe. A mild injury seldom caused pulp necrosis. A moderate injury typically caused pulp canal obliteration. And a severe injury often provoked pulp necrosis.

There is usually no need for endodontic treatment in cases of mild injury because the pulp is likely to survive undisturbed. However, in cases in which the canal space



**Figure 5-18** **A**, 18-year-old male with a history of severe luxation of maxillary left lateral incisor 10 years previously. The tooth shows a complete pulp calcification. **B**, Clinically, the tooth shows a yellowish discoloration of the crown.

begins to calcify, it has been questioned if endodontic treatment is necessary as a “preventive” measure.<sup>8,34</sup> Investigations<sup>50,51</sup> have evaluated teeth with calcified (obliterated) canal spaces after luxation injuries. They found that just over half of these teeth responded normally to pulp testing at the end of the observation period (7 to 22 years). Furthermore, 40% of these teeth did not show any clinical or radiographic signs of pulp necrosis. When compared with normal intact teeth, there was not a higher frequency of pulp necrosis in teeth with calcified canals, even when these teeth were subjected to subsequent caries, new trauma, orthodontic movement, or crown coverage.<sup>8,50</sup> For these reasons, prophylactic endodontic intervention on a routine basis does not seem justified. Also observed in this study of calcified canals was an increased frequency of yellow crown discoloration<sup>50</sup> (Fig. 5-18). This was primarily attributed to a lack of translucency caused by the increased thickness of pulp chamber dentin and the fact that tertiary dentin is usually darker in color compared with normal dentin.<sup>50</sup> This yellowish discoloration may cause an esthetic problem that external bleaching will not easily correct. Veneers or full coverage crowns are still the most predictable esthetic treatment options for these teeth. If the pulp of the calcified and discolored tooth becomes necrotic, endodontic treatment will be necessary, and the yellowish discoloration can then be improved with internal bleaching.

A rare pulpal reaction to trauma is internal root resorption (Fig. 5-19). It has been reported to occur in only about 2% to 3% of all traumatic injuries.<sup>10</sup> To properly diagnose this, a minimum of two periapical radiographs are necessary, whereby the radiographs are exposed with varied horizontal angulations (mesial and distal) (Fig. 5-20). If the resorptive defect presents the same radiodensity as the root canal space and stays located in the center of the tooth in both opposite angulations, then it is likely that the defect is internal root resorption. These resorption lesions expand at the expense of the canal and tend to be symmetrically located within the root canal space.<sup>6</sup> Histologically the pulp tissue seems to undergo a transformation such that it starts resembling granulation tissue, with giant cells resorbing the internal walls of the root.<sup>5</sup> The treatment recommendation is to initiate root canal therapy as soon as possible. Once the blood supply to the main pulpal tissue is removed by the endodontic treatment, the resorptive tissue will cease to proliferate, providing that it has not broken through the lateral walls of the root.

### Periradicular

The periradicular changes subsequent to luxation injuries can be divided into two main categories:

- Periapical changes
- External root resorption



**Figure 5-19** Patient with a history 2 years ago of trauma to the right maxilla. In the lateral incisor, note the extensive intra-canal root destruction secondary to internal root resorption.

The periapical changes are in essence identical to those resulting from any other nonvital and infected root canal system (Fig. 5-21).

In the case of external root resorption, three changes may potentially occur:

- Surface root resorption
- Inflammatory root resorption
- Replacement root resorption

Since these sequelae are typically found following avulsions, they will be discussed in Chapter 6.

Transient apical breakdowns (TAB) have been described in the literature.<sup>4,15</sup> These phenomena are thought to happen irrespective of the injury force.<sup>4</sup> In a case report of a subluxation injury,<sup>15</sup> clear changes consistent with periapical pathosis were observed on follow-up radiographs, yet the teeth responded to vitality (sensitivity) testing. After 10 months, the periapical “lesions” healed without any treatment; however, this is just one case report.

## DIAGNOSTIC MEANS TO DETECT SEQUELAE ON FOLLOW-UP

In dental trauma, radiographs are probably the most important diagnostic tool for assessing healing, particularly if the patient is asymptomatic and the clinical examination does not imply the existence of pathosis. Therefore, every ra-

diograph needs to be of the highest quality and taken with minimal distortion, so that even the subtlest changes can be detected over time. Radiographs must be comparable, reproducible, and reveal not only the root apex, but also the whole contour of the root and associated bone. Several radiographs taken from different angulations are usually necessary to achieve these objectives, both initially and at the follow-up visits.

Pulp evaluation using cold and/or electric pulp tests (EPT) should be performed on the injured tooth and on the adjacent and opposing teeth. The results of this initial pulp testing may prove misleading though. It has been shown in a substantial number of traumatic injuries that the teeth are initially unresponsive to pulp testing.<sup>33,53</sup> However, after a few weeks and depending on the type of trauma, their response can return to normal.<sup>33,53</sup> For this reason, unresponsive teeth should be retested after 8 to 10 weeks or longer to check if vitality has been reestablished. Additionally, teeth with immature pulps frequently do not respond to pulp sensitivity testing.<sup>33</sup> This is the reason why *immature* teeth should be carefully monitored for the next several weeks or months, not only through pulp testing, but also through meticulous clinical and radiographic examinations.

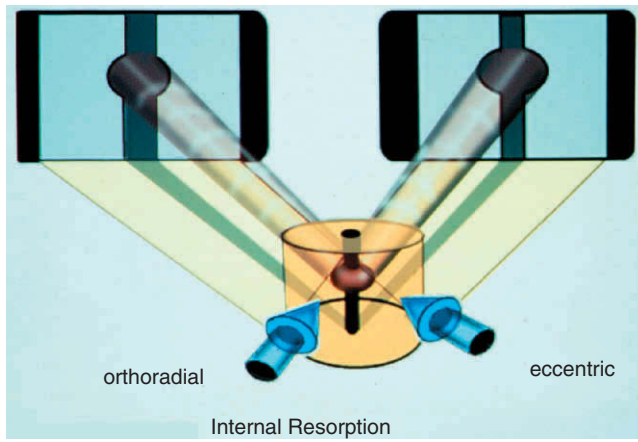
Laser Doppler flowmetry technology has been shown to work reliably and may detect pulp survival and revascularization in a matter of only a few days or weeks posttrauma. This is of extreme interest, as sometimes it is necessary for the clinician, and for the patient, to withstand uncertainty for up to several months or a year before a tooth eventually regains sensitivity or shows root development. Laser Doppler machinery presents the inconvenience of being extremely technique sensitive and expensive (see Chapters 2 and 6).

## EXPECTED HEALING OUTCOME OF LUXATION INJURIES

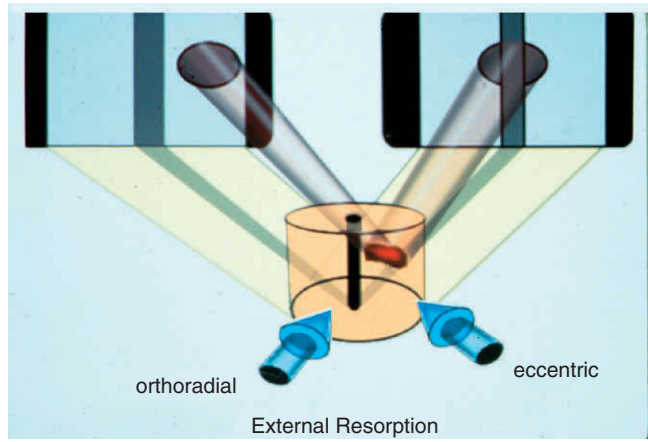
The successful outcome of treating a luxated tooth is that it remains asymptomatic, responds normally to vitality tests, has an intact lamina dura, shows no signs of root resorption, and in the case of an immature tooth continues to develop. However, posttraumatic healing complications may occur, and the clinician must decide when and how to intervene. These complications include one or more of the following signs and/or symptoms:

- Swelling
- Periapical radiolucency
- Sensitivity to palpation
- Sensitivity to percussion
- Increased tooth mobility
- Deep periodontal probing





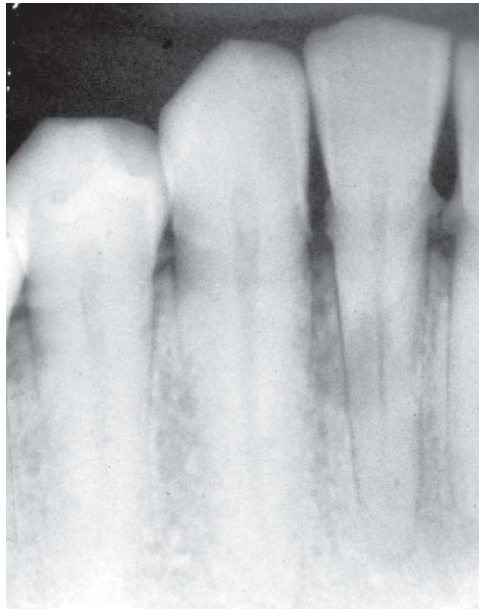
A



B



C



D



E



F

**Figure 5-20** **A** and **B**, Radiographs exposed from two different horizontal angulations aiming to diagnose internal vs. external root resorption. (Courtesy Dr. C. Barthel.) **C** and **D**, This resorption is external because the resorptive defect moves in the opposite direction of the cone. **E** and **F**, This resorption is internal because the defect remains centered in the root irrespective of the angulation.



**Figure 5-21** Nonvital maxillary lateral incisor with a periradicular lesion subsequent to a lateral luxation. Note the absence of caries or restorations, suggestive of trauma as cause of pulp necrosis.

- Bleeding on probing
- Tooth discoloration
- Prolonged nonresponsiveness to pulp vitality (sensitivity) tests
- Spontaneous pain
- Provoked sensitivity or pain to temperature
- Pain on mastication
- Root resorption
- Canal obliteration
- Cessation of root development in the case of an immature tooth

To reinforce patient compliance for the follow-up visits, it is important to make the patient aware of the potential adverse posttrauma sequelae. The clinician must emphasize that certain serious sequelae (such as external inflammatory root resorption) may be asymptomatic, but cause extremely rapid tooth loss if undiagnosed and untreated. Patients should be instructed to contact their dentist immediately if any symptoms develop in the traumatized area.

It is important for the clinician to intervene as soon as unfavorable signs are diagnosed. However, an injured tooth should never be considered nonvital solely because of tooth discoloration or a negative response to pulp testing. It is our recommendation that a tooth be considered nonvital only if a combination of at least two or three signs or symptoms leads

**TABLE 5-2**

**PREVALENCE OF PULPAL NECROSIS AFTER LUXATION INJURY**

| TYPE OF LUXATION   | NUMBER OF TEETH | NUMBER OF NECROTIC PULPS |
|--------------------|-----------------|--------------------------|
| Concussion         | 178             | 5 (3%)                   |
| Subluxation        | 223             | 14 (6%)                  |
| Extrusive luxation | 53              | 14 (26%)                 |
| Lateral luxation   | 122             | 71 (58%)                 |
| Intrusive luxation | 61              | 62 (85%)                 |

From Andreasen FM, Pedersen BV: Prognosis of luxated permanent teeth—the development of pulp necrosis, *Endod Dent Traumatol* 1:207, 1985.

to that diagnosis. For example, in a study<sup>33</sup> monitoring the reason why endodontic therapy was instituted on traumatized teeth, it was found that the most common reason for treatment was a negative response to vitality testing and adverse radiographic changes (73%) or a negative response to vitality testing and discoloration (41%). Discoloration and periapical lesions were found to be the most important diagnostic factors in cases of subluxation, and extrusive and lingual luxations. In this same study, pulp necrosis was diagnosed within 4 months in the 117 teeth evaluated (87%). Diagnoses based on negative vitality tests and discoloration were made within 6 days to 3 months in all but one case.<sup>33</sup>

Table 5-2 shows another study<sup>7</sup> that compared the prevalence of pulp necrosis according to the type of luxation injury. In this study, the investigators did not distinguish between lateral luxation with or without apical displacement; this study clearly shows that an increase in the severity of the injury correlated with less pulp survival.

The stage of root formation plays a important role on pulp survival. Young teeth and teeth with an open apex have the potential to regrow and revascularize pulp tissue within the root canal.<sup>3</sup> It has been estimated that a luxated tooth with an open apex has a 50% greater chance of pulp survival compared with a similar injury in a tooth with a closed apex. Even in cases with moderate intrusive luxations, 45.5% of the teeth remained vital if they had an open apex, compared with no pulp survival if the apex was closed and the tooth had sustained a similar injury.<sup>18</sup>

Concerning progressive external root resorptions (inflammatory and replacement resorption), their frequency following luxation injuries varies between 1% and 47%.<sup>7,11,44,53,55</sup> Furthermore, several studies have shown that their prevalence increases according to the severity of the luxation injury (i.e., progressive external root resorption is less prevalent in concussion injuries [0%] than in intrusion injuries [24%]).<sup>7,11,44,53,55</sup>

These studies are helpful in the sense that they give some guidance to the clinician as to the expected healing outcome of luxated teeth.

Frequent evaluations of luxation injuries are essential. A precise diagnosis of the injury will guide the clinician towards the early detection of subtle signs of postinjury complications.

## CONCLUSION

- After a traumatic episode, even if apparently minor, patients should always be examined as soon as possible and at follow-up visits, as some luxation injuries frequently do not show problems initially.
- Luxation injuries always provoke some degree of damage to the tooth's pulpal neurovascular supply, to its cementum and PDL. These injuries may heal or may cause complications.
- The most frequent complications of luxation injuries include pulp necrosis, pulp canal calcification (obliteration), and external root resorption (surface, inflammatory, and replacement resorption).
- Adverse healing of luxation injuries is related to the severity of the injury, particularly if inappropriate emergency treatment is given.
- The prognosis of luxated teeth is directly related to the severity of the injury and to the stage of root development.
- An exact and precise diagnosis of the injury is of paramount importance because it will guide the clinician towards an accurate and early diagnosis of potential future pathosis.
- The pulp of *immature* luxated teeth should be given a chance to survive, heal, or revascularize. These teeth should be carefully monitored, and they require no endodontic treatment to be performed right away. Endodontic treatment may be necessary in the future if pulp necrosis develops.
- Endodontic therapy is not part of the emergency treatment of luxated teeth and should *never* be performed at the time of the emergency visit.
- The pulp should never be considered necrotic solely because of a single negative response to cold testing or EPT.

Steps for the management of displaced-luxated teeth (laterally luxated, extruded, and intruded):

- A. Emergency visit
  1. Reposition the tooth.
  2. Place a semiflexible splint, if indicated.
  3. Suture soft tissue lacerations, if indicated.
  4. Give appropriate prescriptions and recommendations to the patient.

5. Schedule a reevaluation appointment at 7 to 10 days posttrauma.
- B. Appointment at 7 to 10 days posttrauma
  1. Check soft tissue healing.
  2. Perform endodontic evaluation and/or treatment.
  3. Remove the splint if there is no associated bone fracture.
- C. Reevaluate with subsequent visits and perform necessary treatment if adverse healing is detected.

## REFERENCES

1. Andreasen FM: Pulpal healing following acute dental trauma: clinical and radiographic review, *Pract Proced Aesthet Dent* 13:315, 2001.
2. Andreasen FM: Pulpal healing after luxation injuries and root fracture in the permanent dentition, thesis, Copenhagen, 1995, Copenhagen University.
3. Andreasen FM: Pulpal healing after luxation injuries and root fracture in the permanent dentition, *Endod Dent Traumatol* 5:111, 1989.
4. Andreasen FM: Transient apical breakdown and its relation to color and sensibility changes after luxation injuries to teeth, *Endod Dent Traumatol* 2:9, 1986.
5. Andreasen FM, Andreasen JO: Diagnosis of luxation injuries: the importance of standardized clinical, radiographic and photographic techniques in clinical investigations, *Endod Dent Traumatol* 1:160, 1985.
6. Andreasen FM, Andreasen JO: Luxation injuries. In Andreasen JO, Andreasen FM, editors: Textbook and color atlas of traumatic injuries to the teeth, Copenhagen, 1994, Munksgaard Intl.
7. Andreasen FM, Pedersen BV: Prognosis of luxated permanent teeth—the development of pulp necrosis, *Endod Dent Traumatol* 1:207, 1985.
8. Andreasen FM, Zhijie Y, Thomsen BL et al: Occurrence of pulp canal obliteration after luxation injuries in the permanent dentition, *Endod Dent Traumatol* 3:103, 1987.
9. Andreasen JO, Andreasen FM, Skeie A et al: Effect of treatment delay upon pulp and periodontal healing of traumatic dental injuries, *Dent Traumatol* 18:116, 2002.
10. Andreasen JO: Etiology and pathogenesis of traumatic dental injuries. A clinical study of 1298 cases, *Scand J Dent Res* 78:339, 1970.
11. Andreasen JO: Luxation of permanent teeth due to trauma. A clinical and radiographic follow-up study of 189 injured teeth, *Scand J Dent Res* 78:273, 1970.
12. Andreasen JO, Hjorting-Hansen E: Intraalveolar root fractures: radiographic and histologic study of 50 cases, *J Oral Surg* 25:414, 1967.
13. Application of the international classification of diseases and stomatology, IDC-DA, ed 3, 1992, WHO.
14. Borssen E, Holm AK: Traumatic dental injuries in a cohort of 16-year-olds in northern Sweden, *Endod Dent Traumatol* 13:276, 1997.
15. Boyd KS: Transient apical breakdown following subluxation injury: a case report, *Endod Dent Traumatol* 11:37, 1995.
16. Bystrom A, Claesson R, Sundqvist G: The antibacterial effect of camphorated paramonochlorophenol, camphorated phenol and calcium hydroxide in the treatment of infected root canals, *Endod Dent Traumatol* 1:170, 1985.



17. Chantawiboonchai P, Iida J, Soma K: Effects of aging on oxytalan fibre in mouse periodontal ligament, *J Med Dent Sci* 46:75, 1999.
18. Chaushu S, Shapira J, Heling I et al: Emergency orthodontic treatment after the traumatic intrusive luxation of maxillary incisors, *Am J Orthod Dentofacial Orthop* 126:162, 2004.
19. Crona-Larsson G, Bjarnason S, Noren JG: Effect of luxation injuries on permanent teeth, *Endod Dent Traumatol* 7:199, 1991.
20. Crona-Larsson G, Noren JG: Luxation injuries to permanent teeth—a retrospective study of etiological factors, *Endod Dent Traumatol* 5:176, 1989.
21. Cunha RF, Pavarini A, Percinoto C et al: Influence of surgical repositioning of mature permanent dog teeth following experimental intrusion: a histologic assessment, *Dent Traumatol* 18:304, 2002.
22. Cvek M: Treatment of nonvital permanent incisors with calcium hydroxide. II. Effect on external root resorption in luxated teeth compared with effect of root filling with gutta-percha. A follow-up, *Odontol Revy* 24:343, 1973.
23. Ebeleseder KA, Santler G, Glockner K et al: An analysis of 58 traumatically intruded and surgically extruded permanent teeth, *Endod Dent Traumatol* 16:34, 2000.
24. Eklund G, Stalhane I, Hedegard B: A study of traumatized permanent teeth in children aged 7-15 years. Part III. A multivariate analysis of post-traumatic complications of subluxated and luxated teeth, *Sven Tandlak Tidsskr* 69:179, 1976.
25. Faria G, Silva RA, Fiori-Junior M et al: Re-eruption of traumatically intruded mature permanent incisor: case report, *Dent Traumatol* 20:229, 2004.
26. Flores MT, Andreasen JO, Bakland LK et al: Guidelines for the evaluation and management of traumatic dental injuries, *Dent Traumatol* 17:193, 2001.
27. Haapasalo HK, Siren EK, Waltimo TM et al: Inactivation of local root canal medicaments by dentine: an in vitro study, *Int Endod J* 33:126, 2000.
28. Holan G, Ram D, Fuks AB: The diagnostic value of lateral extraoral radiography for intruded maxillary primary incisors, *Pediatr Dent* 24:38, 2002.
29. Holan G, Ram D: Sequelae and prognosis of intruded primary incisors: a retrospective study, *Pediatr Dent* 21:242, 1999.
30. Humphrey JM, Kenny DJ, Barrett EJ: Clinical outcomes for permanent incisor luxations in a pediatric population. I. Intrusions, *Dent Traumatol* 19:266, 2003.
31. Jacobs SG: The treatment of traumatized permanent anterior teeth: case report & literature review. Part I. Management of intruded incisors, *Aust Orthod J* 13:213, 1995.
32. Jacobsen I: Clinical problems in the mixed dentition: traumatized teeth—evaluation, treatment and prognosis, *Int Dent J* 31:99, 1981.
33. Jacobsen I: Criteria for diagnosis of pulp necrosis in traumatized permanent incisors, *Scand J Dent Res* 88:306, 1980.
34. Jacobsen I, Kerekes K: Long-term prognosis of traumatized permanent anterior teeth showing calcifying processes in the pulp cavity, *Scand J Dent Res* 85:588, 1977.
35. Kenny DJ, Barrett EJ, Casas MJ: Avulsions and intrusions: the controversial displacement injuries, *J Can Dent Assoc* 69:308, 2003.
36. Lee JY, Yanpiset K, Sigurdsson A et al: Laser Doppler flowmetry for monitoring traumatized teeth, *Dent Traumatol* 17:231, 2001.
37. Lee R, Barrett EJ, Kenny DJ: Clinical outcomes for permanent incisor luxations in a pediatric population. II. Extrusions, *Dent Traumatol* 19:274, 2003.
38. Lengheden A, Blomlof L, Lindskog S: Effect of immediate calcium hydroxide treatment and permanent root-filling on periodontal healing in contaminated replanted teeth, *Scand J Dent Res* 99:139, 1991.
39. Luz JG, Di Mase F: Incidence of dentoalveolar injuries in hospital emergency room patients, *Endod Dent Traumatol* 10:188, 1994.
40. Mandel U, Viidik A: Effect of splinting on the mechanical and histological properties of the healing periodontal ligament in the vervet monkey (*Cercopithecus aethiops*), *Arch Oral Biol* 34:209, 1989.
41. Musselwhite JM, Klitzman B, Maixner W et al: Laser Doppler flowmetry: a clinical test of pulpal vitality, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 84:411, 1997.
42. Nikoui M, Kenny DJ, Barrett EJ: Clinical outcomes for permanent incisor luxations in a pediatric population. III. Lateral luxations, *Dent Traumatol* 19:280, 2003.
43. Oikarinen K: Tooth splinting: a review of the literature and consideration of the versatility of a wire-composite splint, *Endod Dent Traumatol* 6:237, 1990.
44. Oikarinen K, Gundlach KK, Pfeifer G: Late complications of luxation injuries to teeth, *Endod Dent Traumatol* 3:296, 1987.
45. Oulis C, Vadiakas G, Siskos G: Management of intrusive luxation injuries, *Endod Dent Traumatol* 12:113, 1996.
46. Pileggi R, Dumsha TC, Myslinksi NR: The reliability of electric pulp test after concussion injury, *Endod Dent Traumatol* 12:16, 1996.
47. Rock WP, Grundy MC: The effect of luxation and subluxation upon the prognosis of traumatized incisor teeth, *J Dent* 9:224, 1981.
48. Ravn JJ, Rossen I: Hyppighed af fordeling af traumatiske beskadigelser af tænderne hos Københavnske skolebørn 1967/68, *Tandlægebladet* 73:1, 1969.
49. Robertson A: A retrospective evaluation of patients with uncomplicated crown fractures and luxation injuries, *Endod Dent Traumatol* 14:245, 1998.
50. Robertson A: Pulp survival and hard tissue formation subsequent to dental trauma. A clinical and histological study of uncomplicated crown fractures and luxation injuries, *Swed Dent J Suppl* 125:1, 1997.
51. Robertson A, Andreasen FM, Bergenholtz G et al: Incidence of pulp necrosis subsequent to pulp canal obliteration from trauma of permanent incisors, *J Endod* 22:557, 1996.
52. Sae-Lim V, Wang CY, Trope M: Effect of systemic tetracycline and amoxicillin on inflammatory root resorption of replanted dogs' teeth, *Endod Dent Traumatol* 14:216, 1998.
53. Skieller V: The prognosis for young teeth loosened after mechanical injuries, *Acta Odontol Scand* 18:171, 1960.
54. Sjogren U, Figdor D, Spangberg L et al: The antimicrobial effect of calcium hydroxide as a short-term intracanal dressing, *Int Endod J* 24:119, 1991.
55. Stalhane I, Hedegard B: Traumatized permanent teeth in children aged 7-15 years, *Sven Tandlak Tidsskr* 68:157, 1975.
56. Tronstad L: Root resorption—etiology, terminology and clinical manifestations, *Endod Dent Traumatol* 4:241, 1988.
57. Trope M: Root resorption of dental and traumatic origin: classification based on etiology, *Pract Periodontics Aesthet Dent* 10:515, 1998.
58. Trope M: Clinical management of the avulsed tooth, *Dent Clin North Am* 39:93, 1995.
59. Trope M, Moshonov J, Nissan R et al: Short vs. long-term calcium hydroxide treatment of established inflammatory

- root resorption in replanted dog teeth, *Endod Dent Traumatol* 11:124, 1995.
60. Trope M, Yesilsoy C, Koren L et al: Effect of different endodontic treatment protocols on periodontal repair and root resorption of replanted dog teeth, *J Endod* 18:492, 1992.
61. Turley PK, Joiner MW, Hellstrom S: The effect of orthodontic extrusion on traumatically intruded teeth, *Am J Orthod* 85:47, 1984.
62. Wang X, Puram S: The toughness of cortical bone and its relationship with age, *Ann Biomed Eng* 32:123, 2004.

# AVULSIONS



ASGEIR SIGURDSSON AND CECILIA BOURGUIGNON

## CHAPTER OUTLINE

### ETIOLOGY AND EPIDEMIOLOGY

### KEY ISSUES ASSOCIATED WITH PROGNOSIS AND MANAGEMENT OF AVULSED TEETH

- Pulpal Damage: Comparing Mature and Immature Teeth
- Periodontal Damage

### EMERGENCY MANAGEMENT OF AVULSION INJURIES

#### At the Site of the Injury (Outside the Dental Office)

- Immediate Replantation
- Transport Medium Before Replantation

#### Emergency Management at the Dental Office

- Patient Examination: Clinical and Radiographic Aspects
- Socket Manipulation
- Root Manipulation
  - Extraalveolar Dry Time Less Than 1 Hour or Tooth Stored in a Proper Storage Medium: Closed Apex
  - Extraalveolar Dry Time Less Than 1 Hour or Tooth Stored in a Proper Storage Medium: Open Apex
  - Extraalveolar Dry Time Greater Than 1 Hour: Closed Apex
  - Extraalveolar Dry Time Greater Than 1 Hour: Open Apex
- Splinting
- Endodontic Treatment Issues at the Emergency Visit
- Systemic Treatment

Antibiotics

Analgesics

Instructions to the Patient

Prognosis Assessment at the Emergency Visit

### SEQUELAE OF AVULSION INJURIES

- Consequences of Pulpal Necrosis
- Consequences of Periodontal Damage: External Root Resorption
  - Surface Root Resorption
  - Replacement Root Resorption
  - Inflammatory Root Resorption
  - Cervical Root Resorption

### POSTTRAUMATIC FOLLOW-UP AND MANAGEMENT

- Need for Endodontic Treatment
- Recall Frequency
- Treatment Options for Open Apex Teeth That Failed to Revascularize
  - Apexification
  - One Versus Two Step Apexification
  - Pulpal Regeneration Modalities

### EXPECTED HEALING OUTCOME OF AVULSIONS

- Epidemiological Data
- Signs of Nonhealing
- Treatment Options

### AVULSED PRIMARY TEETH

### CONCLUSION



The term “dental avulsion” describes a clinical situation in which the tooth is completely displaced out of its socket following a traumatic impact. Of all dental injuries, avulsion is by far the most serious because not only are the pulpal blood vessels and nerves severed, but also the periodontal ligament (PDL) is torn and dehydrates during the extraalveolar dry time before the replacement.

For the longest time, the conventional wisdom was that an avulsed tooth was a lost cause. Until the 1960s, it was understood that the replantation of an avulsed tooth was a temporary measure only because the tooth would almost inevitably end up being lost as a result of root resorption.<sup>72</sup> It was in the late 1960s, when ground breaking studies by Dr. J.O. Andreasen and later by Dr. M. Cvek were published, that this old concept was reconsidered.<sup>20,21,38</sup> Those studies showed that an avulsed and replanted tooth could be maintained providing certain treatment steps were performed in a timely fashion. They demonstrated that the five most critical factors affecting the survival of a replanted avulsed tooth are:

1. Time out of the socket (extraalveolar time)
2. Storage medium
3. Splinting type and duration
4. Condition of the pulp and root canal space
5. Stage of root formation

Since then, further research and observations have shed better light on how the prognosis of a replanted avulsed tooth is affected by these critical factors. These are discussed in detail in this chapter.

## ETIOLOGY AND EPIDEMIOLOGY

Compared with other dental injuries, avulsions are a relatively rare occurrence, accounting for 1% to 16% of all dental injuries.<sup>7,8</sup> Like most dental traumas, maxillary central incisors are the most frequently avulsed teeth in the permanent dentition,<sup>62</sup> being the most typically traumatized teeth with sports activities, playing, and automobile crashes being the most frequent causes for this type of injury.<sup>8,17,34,85</sup> It has been reported that the most frequently involved age group is 7 to 10 years, an age group in which the maxillary incisors are only partially formed.<sup>20</sup> However, according to a more recent study, it appears that the average age of the injured individuals is slightly higher, with a mean age closer to 14.<sup>17</sup> This age difference is of importance because at 14 years the incisor’s apices are closed, and consequently pulpal revascularization is highly improbable after the replantation of an avulsed tooth.

## KEY ISSUES ASSOCIATED WITH PROGNOSIS AND MANAGEMENT OF AVULSED TEETH

### PULPAL DAMAGE: COMPARING MATURE AND IMMATURE TEETH

Pulpal necrosis always occurs after an avulsion injury. However, pulp revascularization can take place in teeth presenting an immature apex (i.e., teeth that still have an apical opening larger than 1.1 mm<sup>40,68,95</sup> [see later section on Emergency Management at the Dental Office]). When the apex is fully formed, there is virtually no possibility for the pulp to revascularize. Thus necrotic pulpal tissue should be removed before infection sets in. If that is not done, a significant drop in the tooth’s prognosis results.<sup>36,37,109</sup>

### PERIODONTAL DAMAGE

The survival of an avulsed and replanted tooth is highly dependent on the duration of the *extraoral time* (particularly if the avulsed tooth is left in open air [i.e., *extraalveolar dry time*]) and on the conditions in which the tooth was stored during the extraalveolar time.

If the root surface dries out, there will be a complete dehydration and breakdown of the PDL and of the root’s protective layers (cementum and precementum). When the precementum is lost or altered, the posttraumatic inflammatory response will include multinucleated clastic cells capable of resorbing the exposed dentin.<sup>107</sup>

To prevent desiccation of the root’s surface, the avulsed tooth should be replanted immediately or kept in an appropriate storage medium until replantation is carried out as soon as possible.

## EMERGENCY MANAGEMENT OF AVULSION INJURIES

### AT THE SITE OF THE INJURY (OUTSIDE THE DENTAL OFFICE)

#### Immediate Replantation

The time spent outside of its socket (“extraalveolar time”) is the most critical factor affecting the survival of an avulsed and replanted tooth,<sup>16–19</sup> particularly if the root surface was allowed to dehydrate because the tooth was left in open air (“extraalveolar dry time”). Therefore the aim of treatment should always be to replant the tooth back into its socket as quickly as possible.

Education of the general public on measures to be taken in case of dental avulsions is crucial. Posters like the one created by the International Association of Dental Trauma-

tology (IADT) (Fig. 6-1) should be posted in every dental office, school, and sports facility. Emphasis should be given to attempting to replant the avulsed tooth as soon as possible. Adults should be advised that if the root is heavily contaminated by surface debris, it should not be scraped or wiped, but rather just rinsed off for a few seconds under running water. The gentle management of the root surface is important to preserve all viable PDL and cemental cells remaining. If debris remains, or if no water is available, the tooth should be stored under the tongue (if conditions allow), placed in a cup with saliva, or replanted as is. Careful assessment of the patient's ability to retain the tooth in his/her mouth is essential to preclude any potential for swallowing or aspirating the tooth. If there is suspicion of the tooth being replanted with debris, the tooth can be removed from the socket by the dentist, rinsed off with sterile saline, and replanted again. The person replanting the tooth should be instructed to hold it by the crown and to place it back into the socket with minimal force. The patient can then bite gently on a piece of cloth, a handkerchief, or anything convenient so as to keep the tooth in place until he/she arrives at the dental clinic. However, it must be noted that if some resistance is met during the repositioning, there might be an alveolar bone fracture associated with the avulsion. The person replanting the tooth should then be instructed to avoid forcing the tooth in place, to soak the tooth in milk or saliva, and bring the patient (and the tooth!) to the dental office as soon as possible.

### Transport Medium Before Replantation

If it is impractical to replant the tooth immediately at the site of the injury, care must be taken to store the tooth in proper conditions so as to increase the chances of survival of the PDL cells still attached to the root surface. Suggested<sup>57</sup> transport media are:

- Cell culture media placed in specially designed containers for tooth transportation, like the Save-A-Tooth™ kit (Phoenix-Lazarus, Inc., Pottstown, Pa.) and the tooth rescue box Dento Save™ (Dentosafe BmbH, Iserlohn, Germany) (Fig. 6-2)
- Milk
- Physiological saline

If none of these transport media are available, it has been suggested to place the avulsed tooth in the vestibule of the mouth because it is better than storing it dry or in water.<sup>57</sup> Water should never be recommended as a transport medium because it is hypotonic and has incompatible osmolality to the surviving cells on the root surface.<sup>96</sup> Massive and rapid cell lysis will start within minutes of the tooth being placed in tap water.<sup>28</sup> Placing the avulsed tooth in saliva (after expectorating in a cup) or in the vestibule of the mouth is better than placing it in water because saliva preserves the periodontal cells for some minutes and up to 2 hours.<sup>28</sup> But saliva's osmolality and pH are still far from ideal to the cells. Furthermore, bacteria existing in saliva constitute an additional challenge to the PDL cells' survival.<sup>73</sup> The specialized

storage containers filled either with Hank's Balanced Salt Solution (HBSS), a pH-preserving fluid, or with other tissue culture media are the best transport media assuming they are available. There is a good indication that these media can keep the periodontal ligament cells alive for an extended period of time of up to 12 or even 24 hours in ideal situations.<sup>57,89</sup> Making these specialized containers available to all people at risk should be a priority to the community and to the dentists. However, these specialized containers have the inconvenience of presenting a high cost and a limited shelf life.

If specialized transport media are unavailable, the best alternative is milk. It is usually readily available, will maintain a high percentage of viable PDL cell count for at least 3 hours, and has an osmolality and a pH close to ideal for cells. Additionally, pasteurized milk is relatively free of bacteria.<sup>28</sup> Milk refrigeration seems unnecessary to maintain cell vitality, even though cooling these cells is not detrimental.<sup>57</sup>

The search for an ideal transport medium continues. Recently, a study<sup>78</sup> indicated that an experimental preparation of *propolis*, an antibacterial and antiinflammatory resinous bee-hive product, was better in maintaining cell viability than saline, milk, or HBSS; at this time it may be premature to recommend this concept.

## EMERGENCY MANAGEMENT AT THE DENTAL OFFICE

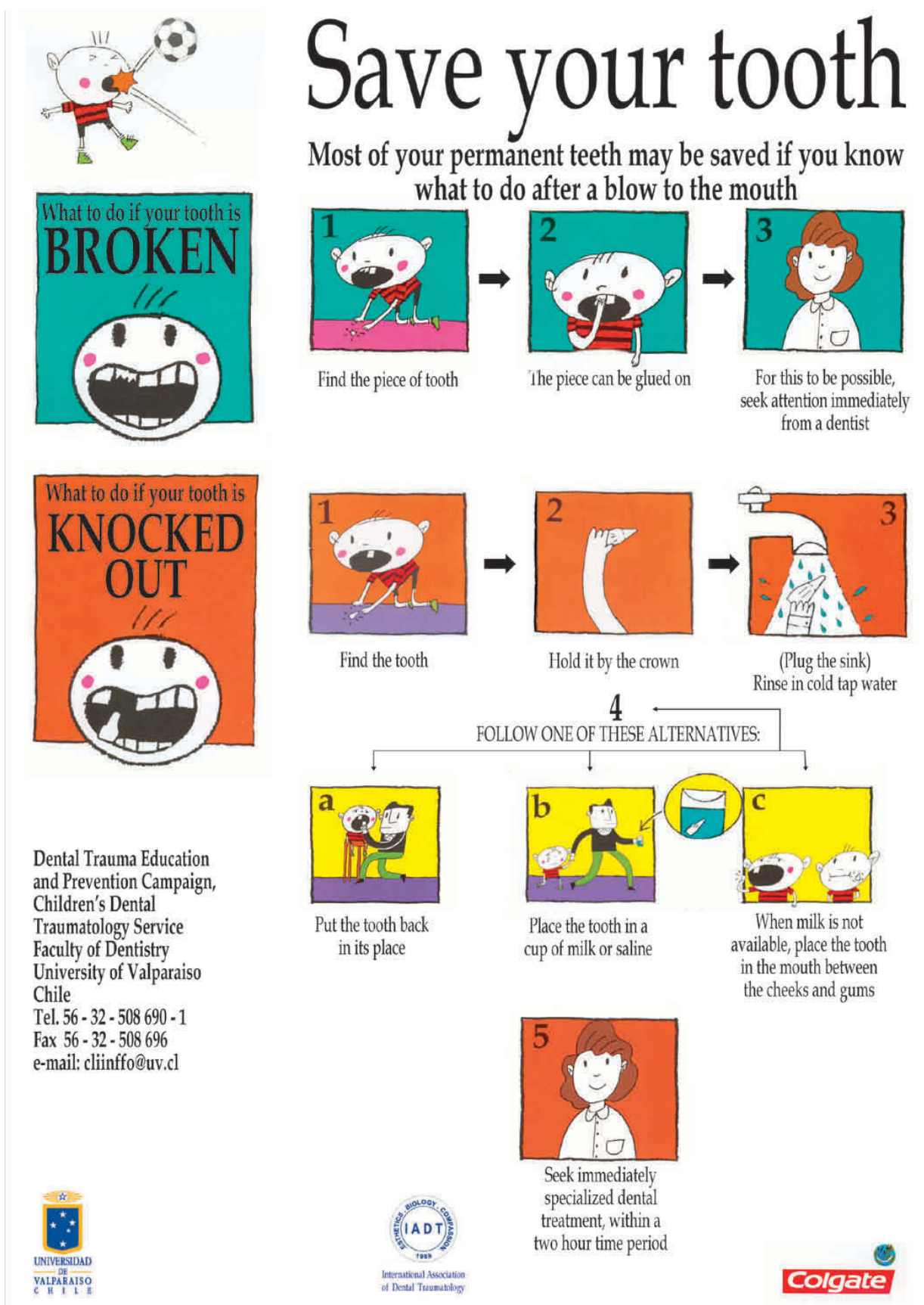
### Patient Examination: Clinical and Radiographic Aspects

The most obvious clinical presentation following an avulsion is an empty socket (Fig. 6-3). However, if the tooth was replanted before arriving at the dental clinic, it will be loose in its socket and in most cases will have the tendency to drift out of its socket.

If the tooth was not replanted before the patient's arrival at the dental office, then its storage condition needs to be immediately assessed upon arrival. If it was stored in an appropriate storage medium, then it should be left in it while all the necessary paperwork and clinical evaluations are done. If, however, it was not stored in proper conditions, then it should be placed immediately in the best available specialized storage medium. Should this be unavailable, the avulsed tooth should be placed in milk or saline before any further evaluation of the patient.

It is important to remember that an impact capable of causing an avulsion is likely to be strong enough to affect other adjacent structures as well. Therefore a full assessment of the site of the injury should be made along with an evaluation of the adjacent and opposing teeth (see Chapter 2).

It is important to expose periapical radiographs of the injured area when the patient arrives at the clinic, irrespective of whether the tooth was replanted or not. This ensures that no portion of the root has been left in the socket and that the tooth has been completely avulsed rather than

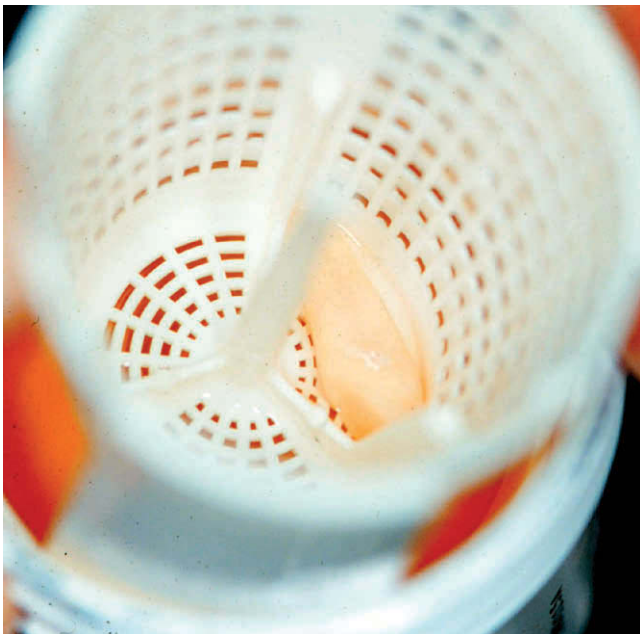


**Figure 6-1** Poster aimed at children and teenagers containing simple instructions on the emergency treatment of dental injuries. (Courtesy the International Association of Dental Traumatology [IADT].)





A



B

**Figure 6-2** **A**, Save-A-Tooth™ system. The sealed container is half filled with HBSS. **B**, The tooth is immersed into the solution by a suspended basket. (A Courtesy Phoenix-Lazerus, Inc., Pottstown, Pa.)

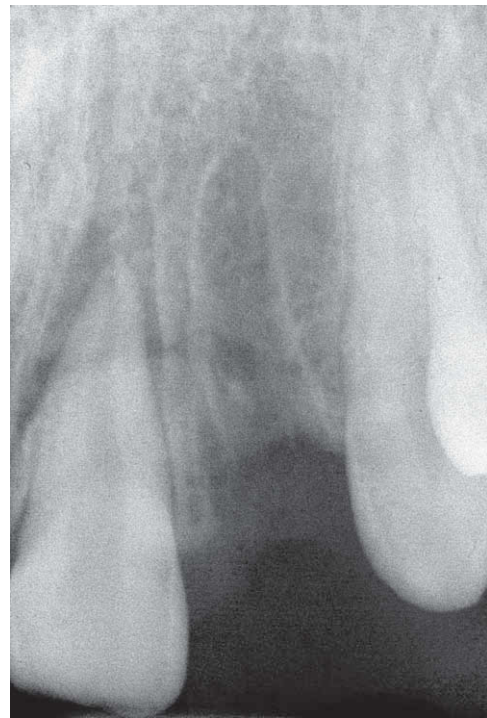
completely intruded. Given the traumatic cause of the avulsion, adjacent and opposite teeth should also be carefully examined radiographically to rule out luxations, root fractures, or other injuries. Multiple radiographs taken from different angulations might be indicated to rule out root fractures of adjacent teeth.

### Socket Manipulation

If some time has elapsed after the injury, the socket is likely to be filled with a blood clot (Fig. 6-4). Replanting the tooth into a coagulum has not been shown to be detrimental to



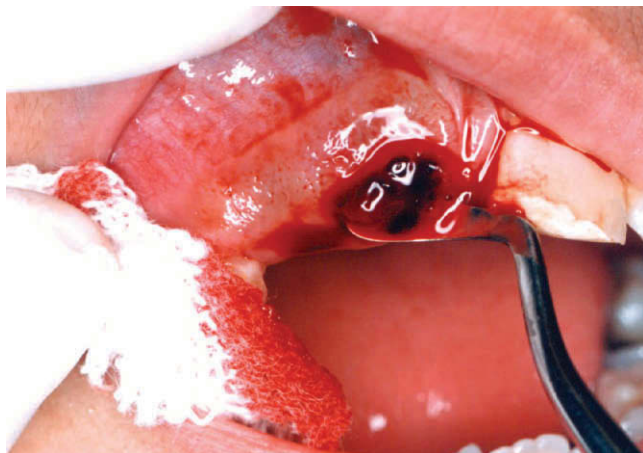
A



B

**Figure 6-3** **A**, A fist-fight injury in which the lateral incisor was avulsed and the central incisor was laterally and extrusively luxated. **B**, Empty socket of avulsed lateral incisor; lateral luxation with apical displacement of the central incisor.

healing; however, the clot may impede proper tooth repositioning. It is therefore advisable to gently rinse the socket with sterile saline before attempting the replantation.<sup>11</sup> Once the socket is cleared of the coagulum, it needs to be assessed to make sure that its walls have not collapsed. If they have, a blunt instrument, such as a mirror handle, should be inserted in the socket and pressure should be applied on the collapsed walls to reposition them. The condition of the socket does not seem to be as important as the condition of



A



B

**Figure 6-4** A, Coagulum in socket should not be curetted. B, Coagulum in socket properly removed by irrigation.

the root surface because studies have shown that the PDL cells of an avulsed tooth tend to remain on the root surface rather than on the socket wall.<sup>59,86,115</sup> Therefore every effort should be made to open up the socket wide enough so that its walls will not cause a scraping injury on the root surface while the avulsed tooth is being inserted back into place.

### Root Manipulation

There are four main treatment choices for an avulsed tooth.<sup>46</sup> The decision on which choice to make is based on the dura-

tion of the extraalveolar dry time and on the stage of root development. If the tooth was kept dry for less than 1 hour, or up to 3 to 4 hours in an appropriate storage medium, then it should be replanted as soon as possible because there is a good likelihood that a reasonable amount of PDL cells on the root surface have survived. If, however, the tooth was kept dry for longer than 1 hour, then there is almost no chance of PDL survival and additional steps need to be taken to slow down the root resorption, which is likely to ensue following the replantation. PDL cells start to die soon after being exposed to drying.<sup>9,12,13</sup> This cell death reaches critical levels after approximately 30 minutes and then increases exponentially after 45 to 60 minutes, up to a point where only a few viable cells remain on the root surface after 60 to 90 minutes in a dry environment.

#### Extraalveolar dry time less than 1 hour or tooth stored in a proper storage medium: closed apex

The tooth should be replanted as soon as possible after the debris has been gently washed off with a stream of sterile saline (Fig. 6-5). Studies<sup>41,87</sup> have investigated soaking a tooth that has been kept dry for more than 20 minutes, but less than 1 hour in HBSS or other tissue friendly media, to determine if this will wash off the dead cells and “revive” the compromised cells. The investigations revealed no better results than replanting the tooth without any previous soaking.

#### Extraalveolar dry time less than 1 hour or tooth stored in a proper storage medium: open apex

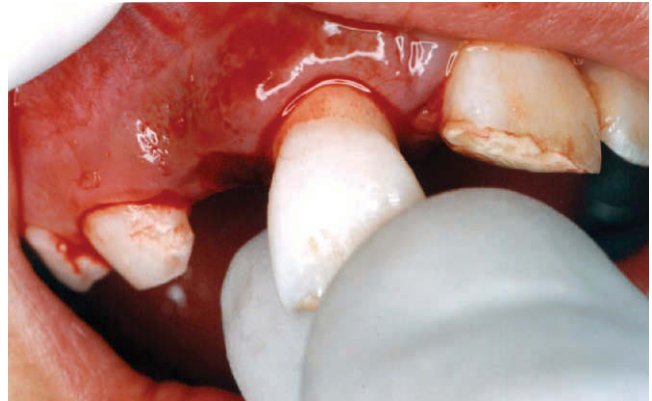
Teeth with an apical foramen larger than 1.1 mm have a potential for pulpal revascularization after replantation<sup>40,68,95</sup> (Fig. 6-6). The rate of revascularization has been shown to vary between 18 and 40%.<sup>40,84</sup> The extraalveolar time and bacterial contamination are additional factors that will influence the rate of revascularization.<sup>68</sup> If bacterial contamination is reduced by soaking the tooth in doxycycline, there is a very significant increase in complete revascularization.<sup>39</sup> Therefore it is now recommended to soak a tooth with an open apex in a solution of doxycycline (1 mg in approximately 10 ml of physiological saline) before replantation. Recent animal studies suggest that covering the root with minocycline (available as Arestin Microspheres™, OraPharma, Inc, Warminster, Pa.) will even further aid in revascularization compared with a doxycycline soak<sup>90</sup> (Fig. 6-7, A and B).

Even though the term “pulp revascularization” is used to name this phenomenon, in many cases the pulpal canal space ends up being filled in its periphery with a nonspecific tissue, which forms some kind of “reactive dentin,” and in its center it becomes partially or even completely filled with a hard tissue that resembles bone rather than dentin<sup>90,95</sup> (Fig. 6-7, C to E). This hard tissue formation has been termed *osteodentin* and in many cases will eventually completely calcify the pulpal space. Attempting to do root canal treatment on these teeth can be difficult because of the disappearance of a canal path as the amorphous hard tissue randomly forms.

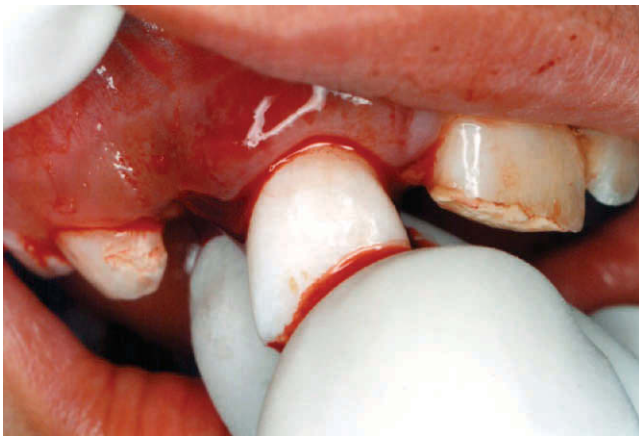




A



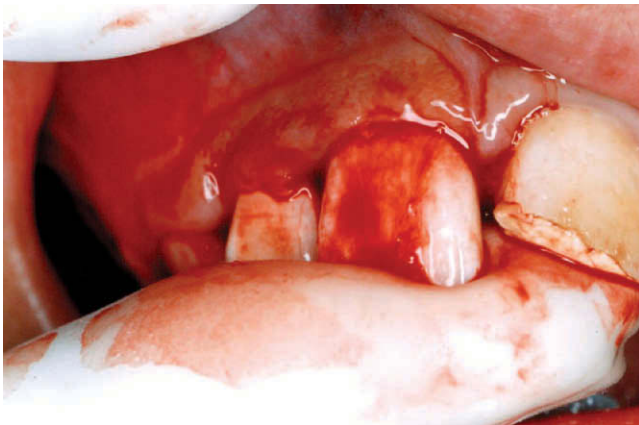
B



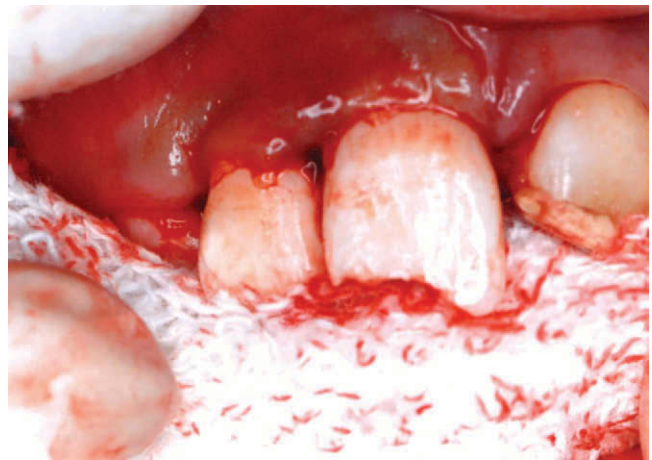
C



D



E



F

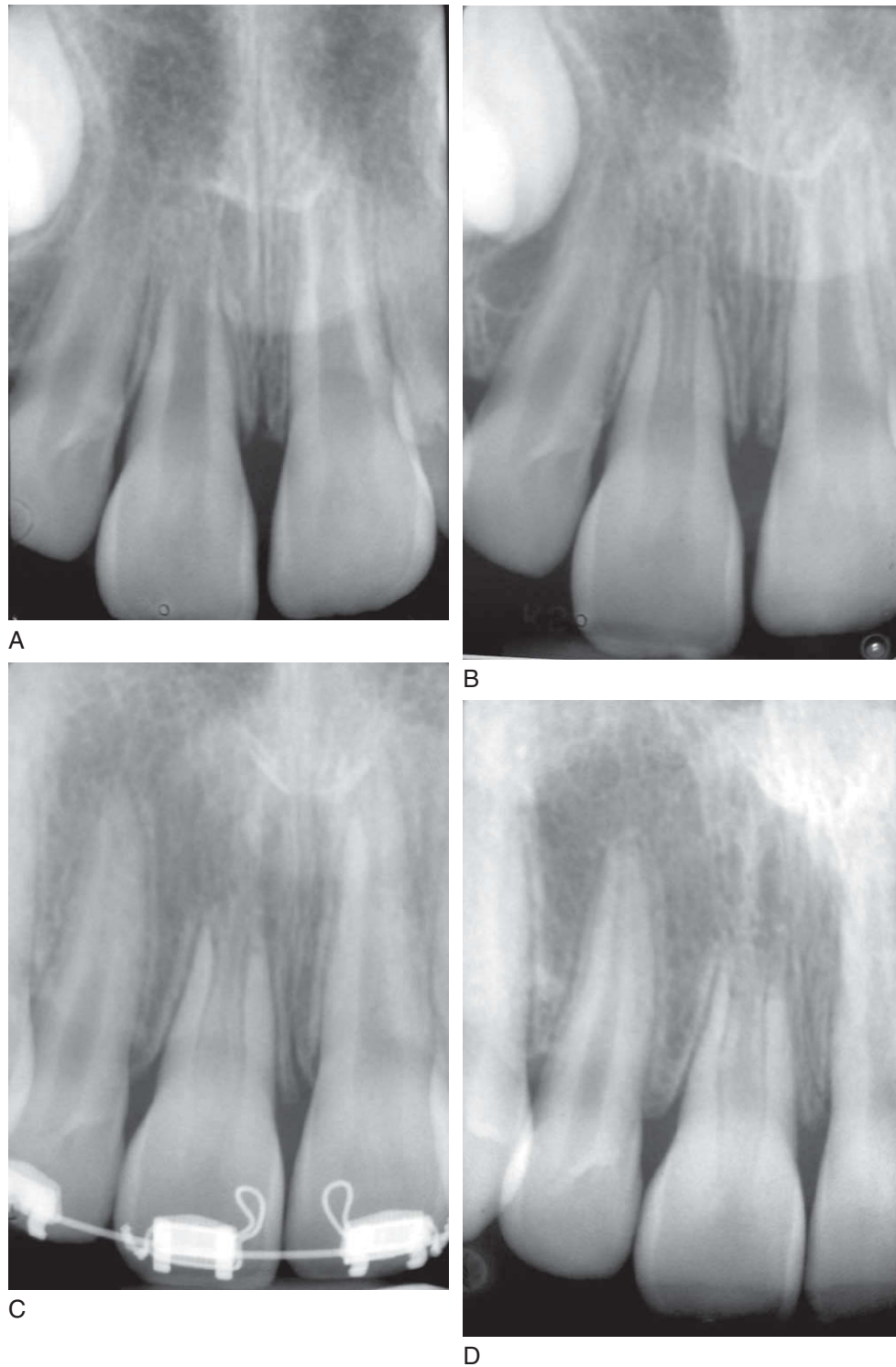
**Figure 6-5** Replantation of avulsed maxillary right lateral and central incisors. **A**, Two avulsed teeth stored in HBSS. **B**, Insertion of central incisor. **C**, Firm pressure on central incisor to position it apically. **D**, Insertion of lateral incisor. **E**, Firm pressure on central and lateral incisors to position them apically. **F**, Final appearance of replantation.

#### Extraalveolar dry time greater than 1 hour: closed apex

When the tooth has been kept dry for more than 1 hour, there are few to no vital cells left on the root surface. Soaking the tooth in various media has not proved to “revive” the cells.<sup>74,98</sup>

Current recommendations (International Association of Dental Traumatology [IADT] Guidelines and American Association of Endodontists [AAE] Guidelines) include scraping off the dead cell’s layer and the remnants of the PDL and then soaking these teeth in 2% stannous fluoride for 5 minutes





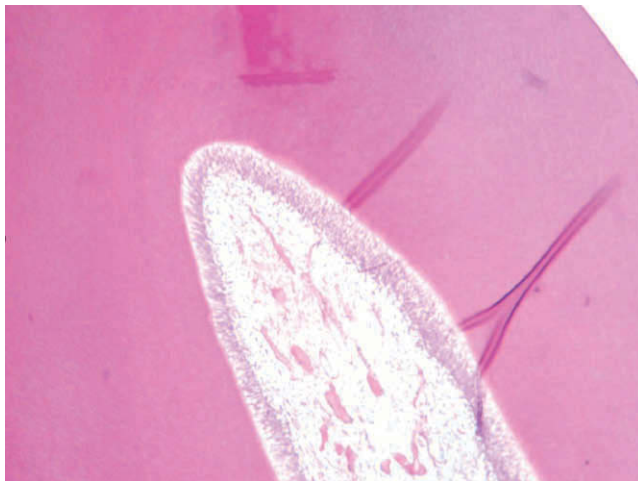
**Figure 6-6** The immature (open apex) maxillary right central incisor was avulsed and replanted after a few hours. The series of radiographs shows bone (or osteodentin) in-growth, which is gradually invading the pulp canal space from an apical to a coronal direction, accompanied by a normal looking PDL. The tooth is not ankylosed and was even successfully moved orthodontically. **A**, One month postreplantation. **B**, Four months postreplantation. **C**, Three years postreplantation with orthodontic treatment in progress. **D**, Twelve years follow-up. The tooth's status is stable.



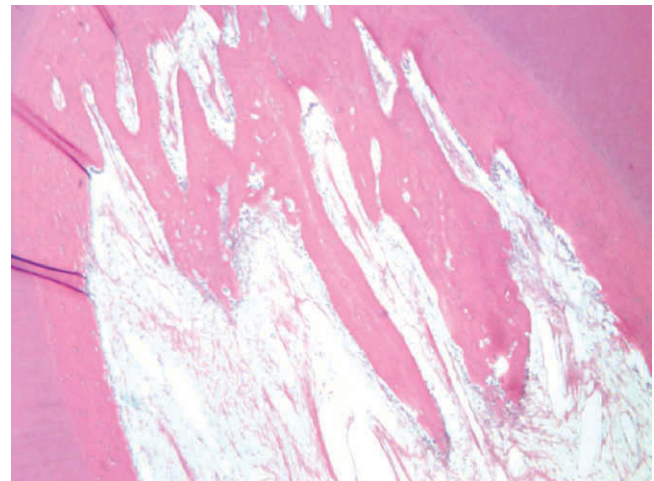
A



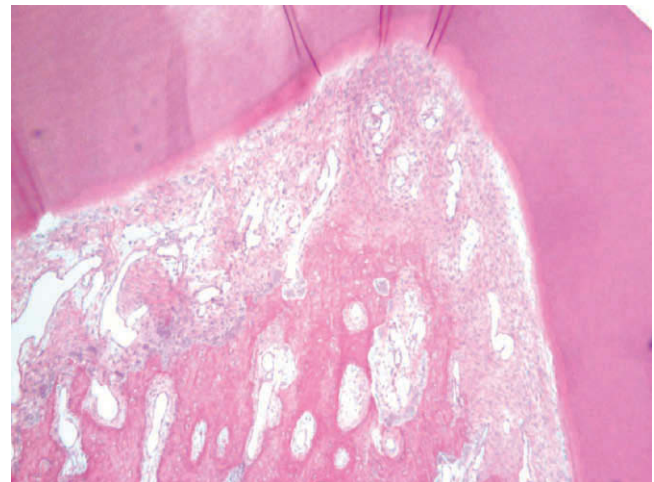
B



C



D



E

**Figure 6-7** **A**, Arestin™. **B**, Covering immature avulsed tooth with Arestin before replantation significantly increases the rate of pulp revascularization compared with no treatment or compared to soaking the tooth in 1 mg/10 ml of doxycycline/saline solution. The following histological sections show different healing patterns of young dogs' pulps after extraction, covering of the root with Arestin™, and replantation. **C**, Normal pulp, with a newly formed but irregular odontoblastic layer. **D**, Normal pulp with a very thick layer of reaction dentin (RD) forming against the dentin existing before extraction and replantation. **E**, Pulp canal space partially filled with "osteodentin," which resembles bone more than dentin and which, in many cases, will fill in the whole pulpal lumen. (Courtesy Dr. A. Ritter.)

before replantation because this will slow down root resorption.<sup>27</sup> However, the prognosis is often poor for teeth with a closed apex and an extraalveolar dry time of more than 1 hour. Some treatment guidelines have recommended against replanting these teeth.<sup>46</sup>

Several encouraging case reports and short duration studies were published a few years ago whereby teeth that had extended extraalveolar dry times were covered with an enamel matrix derivative gel (Emdogain™, Biora/Straumann, Switzerland). The Emdogain™-covered teeth

did not show, at least initially, the massive ankylosis and root resorption like those that had not been covered.<sup>44,45,60,94</sup> Unfortunately, more recent and longer-term studies do not confirm the long-term positive effect of the Emdogain™.<sup>69,94</sup>

#### Extraalveolar dry time greater than 1 hour: open apex

Replantation of teeth with an open apex and extended extraalveolar dry time have the worst prognosis. This is because neither the pulp, cementum, nor PDL have any chance of revascularization or regeneration. It might be beneficial to do the root canal therapy before replanting these teeth because of the difficulty of thoroughly obturating the canal, particularly in its apical portion. Because of the very thin roots, these teeth will eventually end up being lost as a result of ankylosis. If the child is young, there is also a strong possibility that the tooth will become infraoccluded once the alveolus grows.<sup>75,76</sup> However, retaining the tooth may maintain the alveolar ridge, as opposed to an early extraction and subsequent bone loss. In these cases, a *decoronation procedure* has been recommended<sup>75,76</sup> whereby the crown of the tooth is removed to the height of the crestal bone and the root maintained submerged under the overlying gingivae (Fig. 6-8). When the child reaches adulthood, a fixed prosthesis can be fabricated.

#### Splinting

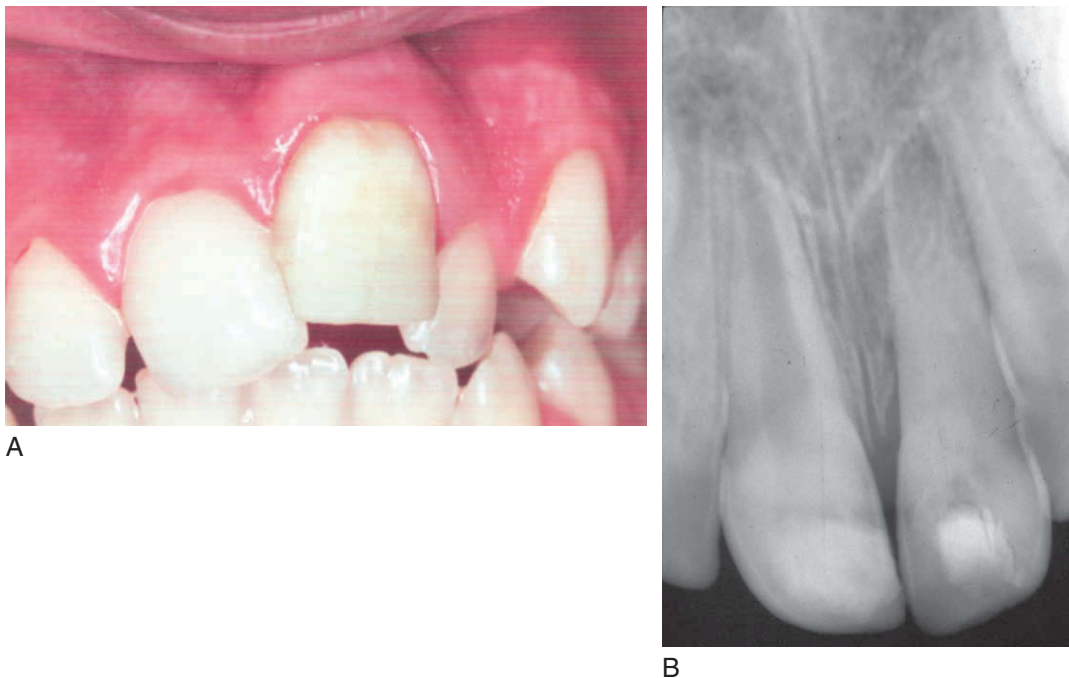
The splinting principles for luxated teeth also apply to avulsed teeth (see Chapter 5). Avulsed teeth must be splinted for 7 to 10 days with a semiflexible physiological splint,

which allows some tooth movement. This type of semirigid splinting minimizes the tendency of the replanted tooth to drift in an incisal direction after replantation. The type of splint seems to be less important on the outcome than the duration of splinting.<sup>19</sup> In monkeys, replanted teeth splinted for thirty days developed a higher incidence of replacement resorption than teeth splinted for only 1 week.<sup>81</sup>

#### Endodontic Treatment Issues at the Emergency Visit

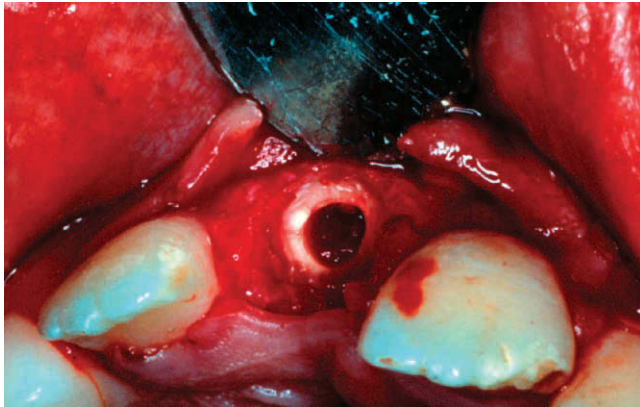
It is not recommended to initiate root canal therapy on an avulsed tooth at the time of injury.<sup>14,22</sup> This is because every effort should be made to minimize the duration of the extraoral time, and performing root canal therapy before replantation would increase this time. Once the tooth has been replanted, it is still loose in its socket, even if well splinted, and drilling the access cavity would be difficult at best and potentially harmful to the PDL.<sup>14,22</sup> Additionally, it has been shown that placing calcium hydroxide too early in the root canal system after replantation might be detrimental to the healing processes of the PDL.<sup>71</sup> Delaying the initiation of the root canal treatment and the placement of the root canal treatment for at least 2 weeks, and the placement of calcium hydroxide is recommended to allow enough time for the PDL to reattach.<sup>14,22</sup>

Recent animal studies have indicated that placing Ledermix Paste™ (Lederle Pharmaceuticals, Wolfrathausen, Germany) immediately into the root canal system of an avulsed tooth, with less than a 60 minute extraalveolar dry



**Figure 6-8** **A**, Avulsion with extended extraoral dry time occurred 8 months before the ankylosis of the maxillary right central incisor in this 10-year-old child, causing severe infraocclusion. There was extended extraoral dry time, so to maintain the volume of bone a decoronation procedure was performed. **B**, Radiograph showing arrested tooth development and ankylosis.

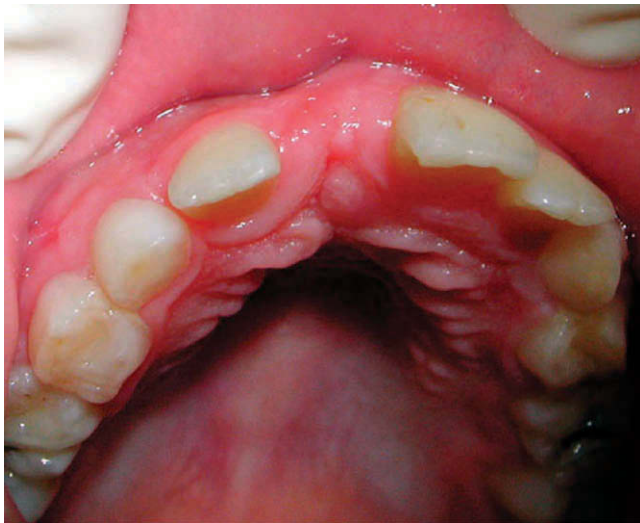




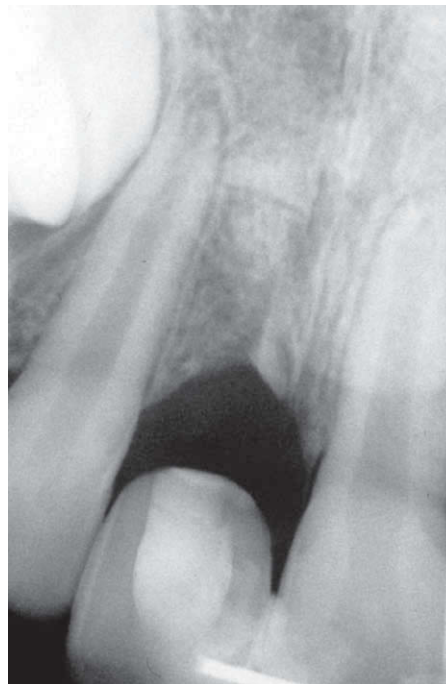
C



D



E



F

**Figure 6-8, cont'd C,** Gingival tissue reflected and the crown removed to the level of the crestal alveolar bone. Root canal filling material was removed and canal space was rinsed with saline; an endodontic instrument was passed through and beyond the apex to create bleeding into the pulp canal space. **D,** Primary closure is achieved by suturing the gingival tissue. **E,** Three-year recall with no collapse of the facial bone. **F,** Most of the remaining ankylosing root left in situ has resorbed and the height of the crestal bone appears to be almost normal.

time, will allow for significantly better healing and less root resorption compared with immediate placement of calcium hydroxide.<sup>29</sup> The Ledermix Paste™ is commercially available and has been used for pulp-capping procedures and for disinfection of the root canal space. It contains triamcinolone and demeclocycline. Triamcinolone is a highly active steroid providing a potent antiinflammatory action; demeclocycline is a broad-spectrum antibiotic effective against a large range of gram-positive and gram-negative bacteria. The Ledermix

Paste™ has demonstrated an antiinflammatory activity that may slow down the root resorption processes after severe traumatic injuries; it is able to diffuse through dentin in concentrations sufficient to sustain effective antiinflammatory levels.<sup>1,3,88</sup> Ledermix Paste™ causes crown discoloration, especially in young teeth.<sup>64,65</sup> To prevent discoloration, the clinician should remove all residual paste from the coronal portion of the access cavity and seal off the paste below the cervical area of the tooth. The steroid content of the

Ledermix Paste™ has raised some concern about its possible systemic effects, even when limited to the root canal space.<sup>2</sup> One study showed that it is unlikely that the amount of steroid used inside the root canal system of teeth would have any systemic effects.<sup>2</sup>

The only indication for performing extraoral root canal treatment following an avulsion is when the teeth have been dry for several hours. In these cases, particularly for immature teeth, endodontic therapy is more conveniently done before replantation. Because of their poor prognosis and high likelihood of becoming ankylosed, some treatment guidelines have suggested not replanting these teeth.<sup>46</sup> This is a case-by-case judgment. If the tooth is not replanted, attempts should be made to maintain the tooth's space and the alveolar ridge until the patient is old enough for an implant or for a fixed restorative appliance. Replanting these teeth may be advisable, even if they serve only as a temporary space and alveolar ridge maintainer.

## Systemic Treatment

### Antibiotics

The administration of systemic penicillin after the replantation of an avulsed tooth has been recommended for the past 20 years. Penicillin's role is primarily to decrease the occurrence of resorption complications, but it does not seem to influence pulpal survival.<sup>52</sup> However, recent follow-up studies on humans have called into question the real benefit of using penicillin or amoxicillin in avulsion cases.<sup>18,19</sup> These studies could not demonstrate any effect on the frequency of pulpal or periodontal healing after taking these antibiotics.

In periodontal research, tetracycline-based antibiotics, especially doxycycline,<sup>56,82,90,93</sup> have been widely used. It has been shown, for example, that systemic doxycycline prevents root resorption and alveolar bone loss in rats after periodontal surgery and that it will improve bone regeneration after periradicular surgery.<sup>35,50</sup> A low dosage of systemically administered doxycycline will reduce orthodontically induced root resorption.<sup>79</sup> In avulsion cases, animal studies reveal that doxycycline given two times a day for a week will significantly lead to more complete healing compared with no antibiotics.<sup>92</sup> Based on this, the International Association of Dental Traumatology (IADT) recommends the use of doxycycline at the appropriate dosage for age and weight, twice a day for 1 week after the tooth has been replanted.<sup>46</sup> Long-term systemic exposure to tetracycline-like medications will affect the forming dentin and possibly cause its discoloration.<sup>47</sup> Therefore it is not recommended to prescribe doxycycline to children under the age of 10 or to those slightly older who have anterior teeth only partially developed. Instead, these children should be prescribed (depending upon body weight and no allergies) 500 mg of penicillin 4 times a day for 7 days. Special precautions should also be taken not to give tetracycline-based antibiotics to women who are pregnant or nursing.<sup>47</sup>

### Analgesics

Appropriate analgesics should be recommended and/or prescribed as necessary. In most cases, nonsteroidal anti-inflammatory drugs such as ibuprofen will suffice.<sup>83,110,111</sup> Although ibuprofen has been shown to be an inhibitor of alveolar bone resorption,<sup>113</sup> no published study has demonstrated its effects on resorption of avulsed teeth.

### Instructions to the Patient

The need for good oral hygiene should be emphasized to the patient at the end of the emergency appointment. However, pain, the presence of the splint, and the fear of causing additional harm may make the patient reluctant or even unable to effectively brush and floss the affected area. This poses a problem because plaque buildup in the sulcus will affect periodontal healing and further compromise the tooth.<sup>117</sup> For this reason, mouth-rinsing with 0.12% chlorhexidine should be recommended twice a day for at least 1 week or as long as the splint is in place.

Additionally, tetanus prophylaxis should be checked and a booster recommended, if necessary (see Chapter 2).

### Prognosis Assessment at the Emergency Visit

The dentist must discuss with the patient and/or his parents the likely prognosis of the teeth involved. It is necessary to emphasize the need for follow-up appointments to ensure the best prognosis. The patient should be aware that even though the crown of the tooth appears normal and in its correct position, it is the root that is the important structure determining the survival of the tooth. The discussion should be realistic and honest. Based on the knowledge of the traumatic circumstances and all of the parameters that occur thereafter, the patient should be informed of the likelihood of the tooth's survival, and care must be taken not to be overly optimistic. Further information about proper informed consent may be found in Chapter 9. Given a fair or poor prognosis, the patient may prefer *not* to replant the tooth, and this should be presented as a treatment option.

## SEQUELAE OF AVULSION INJURIES

### CONSEQUENCES OF PULPAL NECROSIS

The two main consequences of pulpal necrosis are:

- Cessation of root development
- Bacterial contamination of the pulpal space

The bacterial contamination will become the driving force behind destruction of the root and of the surrounding bone if not prevented or addressed in a timely fashion (see below).

## CONSEQUENCES OF PERIODONTAL DAMAGE: EXTERNAL ROOT RESORPTION

Under normal circumstances, permanent teeth do not resorb. This fact appears to be caused by antiresorptive properties of the precementum on the external surface of the root and of the predentin on the internal surface of the root.<sup>107</sup> Initially, after the replantation of a tooth or in the case of a repositioning after a luxation injury, there is a rapid and massive inflammatory response in the PDL. If the damage to the PDL, to the precementum, and to the cementum is minimal and the tooth is replanted within a few minutes, then the PDL will heal without major consequences. This healing occurs in 2 to 4 weeks provided there are no further irritants, such as infection in the pulpal space, to maintain the inflammatory response.<sup>77</sup> However, if there is severe damage to these structures, the root will undergo resorption. The severity and type of root resorption will be directly related to the severity and type of the injury sustained.

### Surface Root Resorption

Surface root resorption probably occurs following all luxation injuries.<sup>25,54</sup> It is the most benign of all resorptions. It is self-limiting provided that no further irritants maintain the inflammation after the initial healing phase. In many cases, this resorption passes unnoticed and undiagnosed because of the difficulty in visualizing it radiographically.

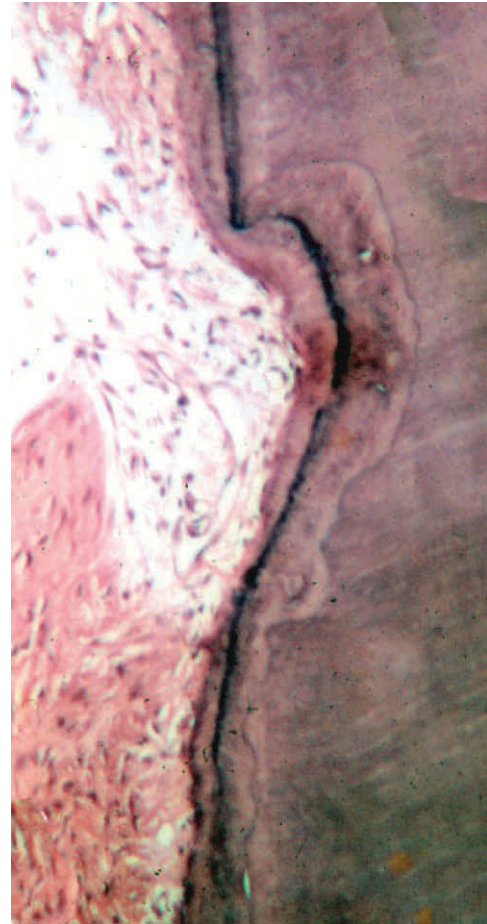
Histologically, areas of disruption of the precementum and cementum are seen. Small and superficial lacunae form into the root surface, but the area is then repaired with new but somewhat irregular cementum (Fig. 6-9). The healing is thought to occur by PDL-derived cells.<sup>6,10,15,22</sup>

No treatment is recommended for this type of resorption; there is no connection between its pathophysiology and pulp health status. Therefore, if some irregular areas are diagnosed radiographically on the root surface, every effort needs to be made to confirm pulp vitality. If the pulp is vital, there is no need for treatment. If the pulp is necrotic, or if there is a question regarding its vitality, then these root surface irregularities are most likely secondary to inflammatory root resorption; this is a much more serious type of root resorption requiring immediate treatment.

### Replacement Root Resorption

Replacement root resorption (RRR) (ankylosis) occurs when there has been massive damage to the PDL and to the cementum following the injury. Such severe damage may occur following an intrusive luxation or an avulsion if the extraalveolar dry time has been greater than 1 hour.

If the PDL and cementum cells do not regenerate in a timely fashion, the root dentin is exposed and the neighboring bone marrow cells repopulate the root surface. These cells behave like osteoblasts, rather than like odontoblasts, because they respond to hormones and cytokines that stimulate bone resorption and guide the physiological processes



**Figure 6-9** Histological slide of the healing phase of surface root resorption. The dentin has not and will not be reconstituted; however, the cementum layer and the periodontal ligament structures will heal-in the defect and eventually reconstruct a normal width of periodontal ligament.

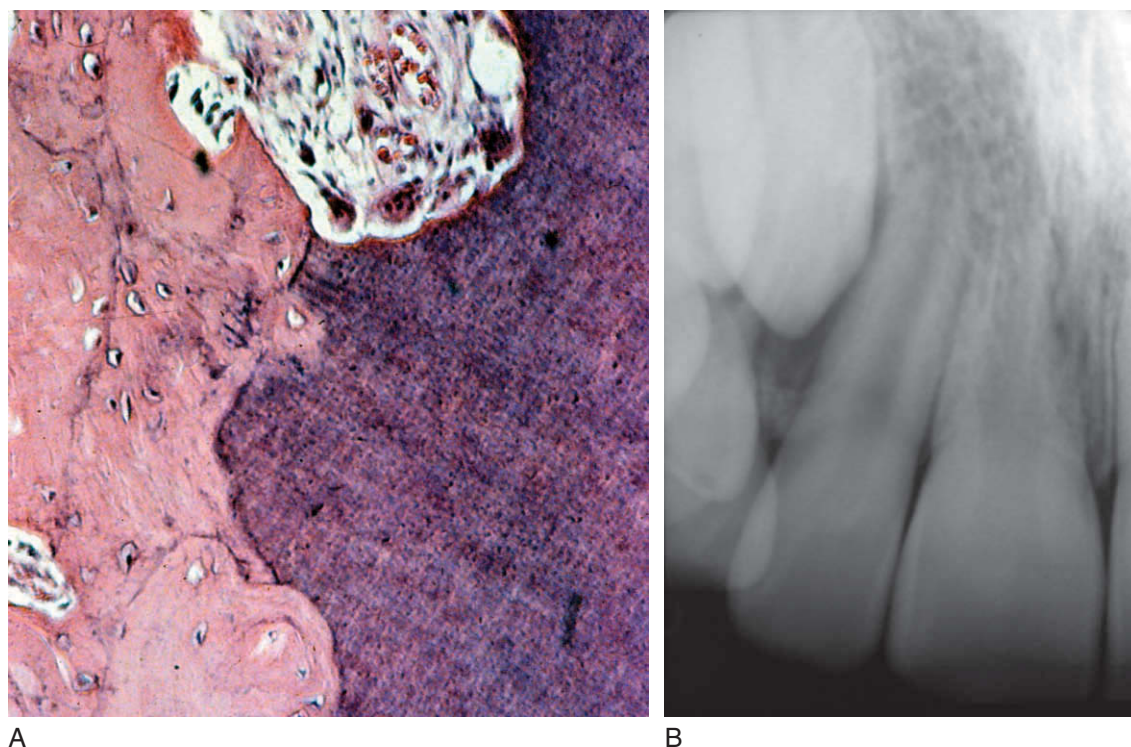
of bone turnover. This is in contrast with odontoblasts, which do not respond to these hormones and cytokines.<sup>53</sup> Thus the root starts to be gradually replaced by bone. For this reason, this type of resorption is termed *replacement resorption*.

There have been two different subtypes of RRR described: *progressive* and *transient* replacement resorption. Progressive replacement resorption occurs when the bone eventually replaces the root completely. Transient replacement resorption occurs when there are relatively small areas of ankylosis that seem to disappear with time.<sup>24</sup> Histologically, there are some areas of fusion between the bone and the dentin, whereas in some other areas there are multinucleated osteoclasts actively resorbing the dentin<sup>20</sup> (Fig. 6-10, A).

Radiographically, the root starts to fade away and no clear PDL space is seen. In advanced stages, the root disappears (Fig. 6-10, B).

RRR can be seen as early as 2 weeks after trauma; but in most cases, it will reveal itself within 2 years after trauma.





**Figure 6-10** **A**, Histological appearance of ankylosis: areas of active root resorption including large multinucleated odonto/osteoclast cells, associated with areas of direct fusion of the alveolar bone with dentin. **B**, Ankylosing immature right maxillary incisor. In some areas, there is a complete loss of the PDL space and the bone appears to fuse with the root.

RRR has been reported to occur up to 10 years post-trauma.<sup>12,19</sup> The high-pitched metallic percussion sound is often the earliest diagnostic sign of ankylosis, and it may be noticed before any resorptive changes are detected radiographically.<sup>5,30</sup>

Currently, there is no treatment available for RRR. These teeth pose a problem for growing children because the fusion of the root to the bone makes the teeth immobile, preventing the teeth from natural maturation with the neighboring teeth and the growing alveolar ridge. As previously described, tooth decoronation and root submersion has been suggested as a temporary measure to maintain the dimensions of the alveolar ridge<sup>75,76</sup> (see Fig. 6-8).

### Inflammatory Root Resorption

Inflammatory root resorption (IRR) occurs when serious trauma affects the cementum and the PDL in such a way that these protective layers are lost in large areas of the root surface (Figs. 6-11 and 6-12), enabling osteo/odontoclastic activity directly on the dentin surface.<sup>25</sup> This activity is amplified if inflammation occurs because of pulp necrosis and infection.<sup>23</sup> When the cemental protective layer is lost, bacteria and bacterial elements, such as endotoxins, have a clear path through the dentinal tubules to the surface of the root. This type of root resorption is usually aggressive in children aged 6 to 10 years because at this age dentinal

tubules are large, and the distance from the pulp canal to the root surface is small.<sup>15,20</sup>

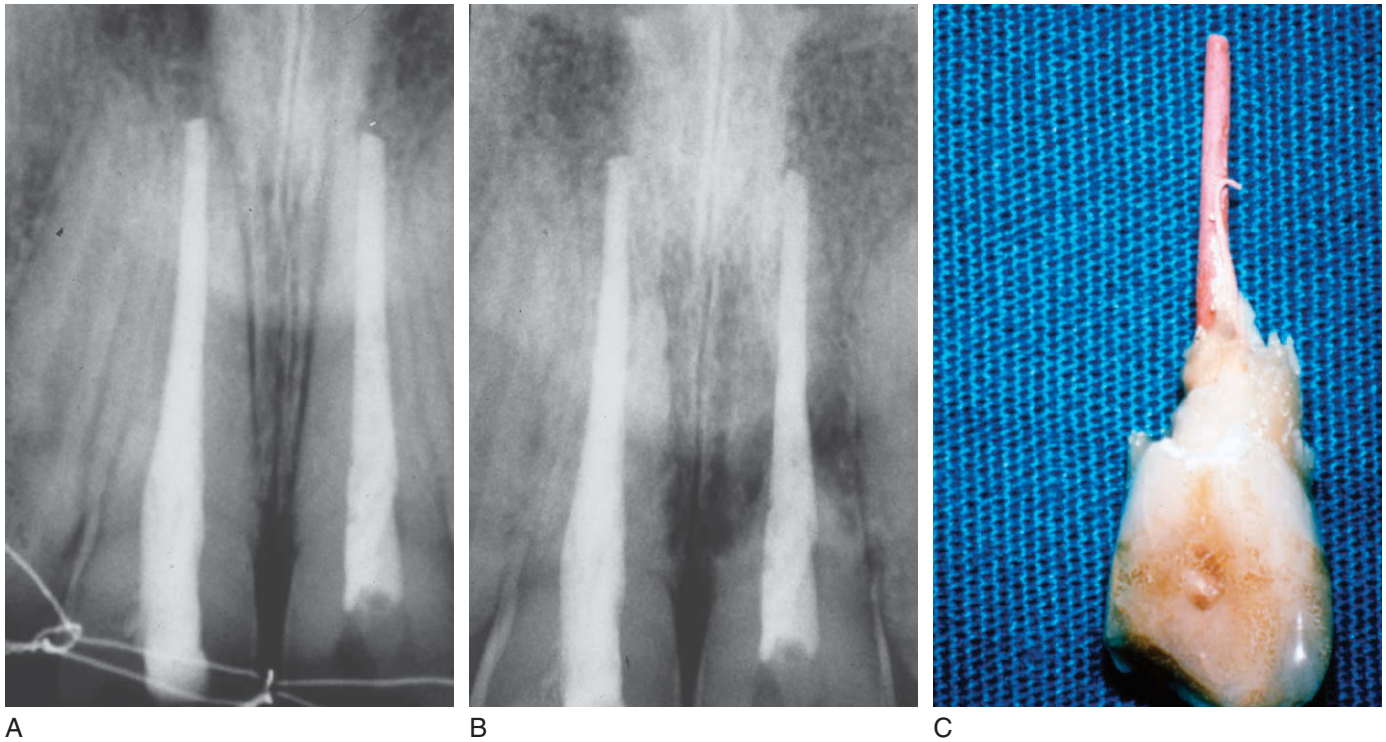
Histologically, there is an infiltration of granulation tissue, with lymphocytes, plasma cells, and polymorphonuclear leukocytes located in root dentin lacunae and in the bone immediately adjacent to where the root resorption is occurring<sup>12</sup> (Fig. 6-13).

Radiographically, IRR may appear initially as a widened and irregular PDL space. Subsequently, distinctive radiolucent lesions will form on the root surface and in the adjacent bone. The first signs of IRR (most commonly located in the cervical third of the root) can be apparent radiographically as early as 2 weeks after the injury, especially in young teeth.<sup>20</sup>

The clinician should be alert for these signs as it may be possible to successfully treat IRR if it is diagnosed early.<sup>33</sup> The key to successful treatment is to completely disinfect the root canal space. Once the canal space is disinfected, the PDL width will return to normal and follow the new contours of the root surface (Fig. 6-14 and see Fig. 6-19).

### Cervical Root Resorption

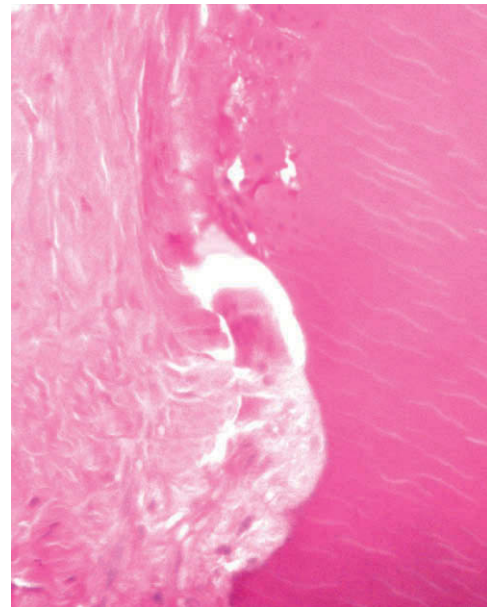
Cervical root resorption (CRR) is a subclassification of IRR appearing immediately below the epithelial attachment of the tooth (Fig. 6-15). It seems to be a delayed reaction to some kind of trauma close to the cemento-enamel junction



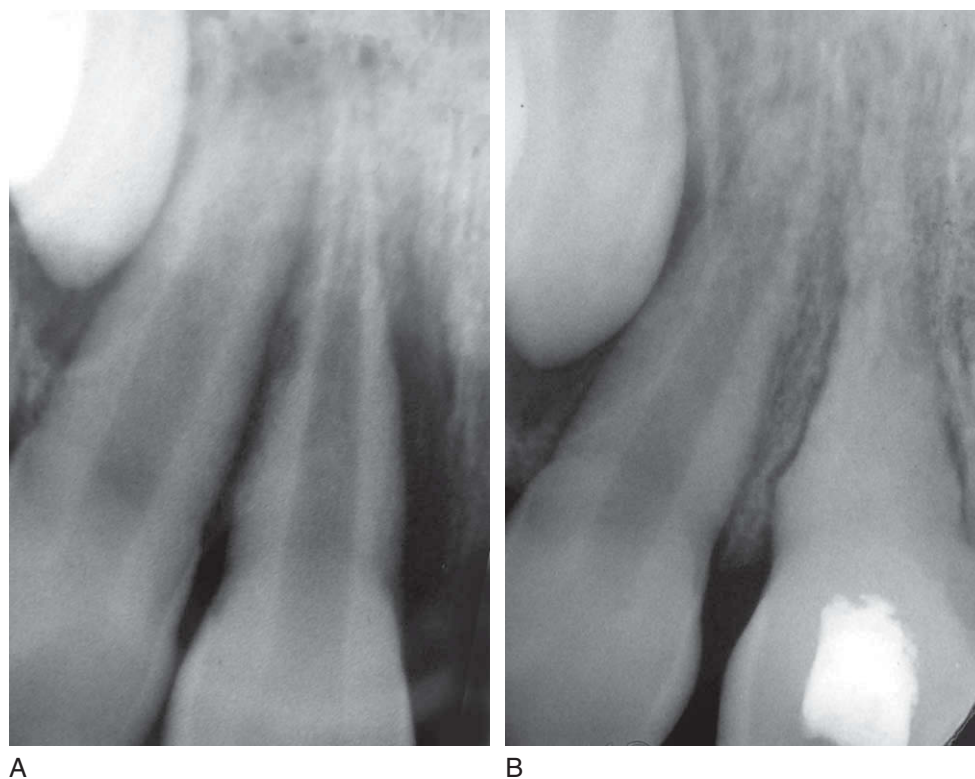
**Figure 6-11** Final stages of progressive replacement resorption, showing some degree of inflammatory resorption associated. **A**, One month after avulsion with extended extraoral dry time. **B**, One year after avulsion with alveolar bone and root resorption, extending to the level of the gutta-percha obturation. **C**, Extracted tooth with root completely resorbed, with gutta-percha remaining.



**Figure 6-12** Radiograph of a tooth presenting with IRR. Note the radiolucent area in the bone associated with the resorptive defect on the root surface.



**Figure 6-13** Histological slide of external IRR. Observe the aggressive granulation tissue including lymphocytes, plasma cells, and polymorphonuclear leukocytes located in lacunas of the root dentin and in the immediately adjacent bone. These inflammatory cells are attracted by the presence of infection within the root canal system.



**Figure 6-14** **A**, Radiograph showing an advanced stage of external IRR. The tooth had been avulsed and replanted. Patient was noncompliant and endodontic treatment was started too late, after much of the root had already resorbed. **B**, Radiograph of the same tooth after being successfully treated with long-term calcium hydroxide therapy. The root surface remains irregular; however, the PDL space has reconstituted itself and presents a normal width that closely follows the root contour irregularities.

or close to wherever the epithelial attachment on the root ends.<sup>106</sup> The exact pathogenesis is unknown, but histologically it resembles IRR with only the location being different. The cause does not seem related to the status of the pulpal space, as CRRs occur equally in vital and nonvital teeth. It has been assumed it is the presence of sulcular bacteria that maintains the lesion once it has formed.<sup>106</sup> CRR can be seen after luxation injuries, orthodontic therapy, bleaching procedures, and periodontal or endodontic surgery.<sup>55,105,106</sup>

Radiographic evaluation will reveal a radiolucent area in the cervical area of the tooth adjacent to the crestal bone (Figs. 6-16 and 6-17). The lesion seems to have the tendency to develop and be confined to the dentin, in an apical/incisal direction along the precementum and predentin, but without perforating these protective layers and without penetrating into the pulp canal space or into the PDL space.

There is no ideal treatment for CRR. Because it seems to be a delayed reaction to the initial trauma, appearing sometimes years after, it is impossible to prevent CRR based upon what is known today. If the pulp is necrotic and infected, endodontic therapy should be provided. However, if the pulp is vital, endodontic therapy will not arrest the progression

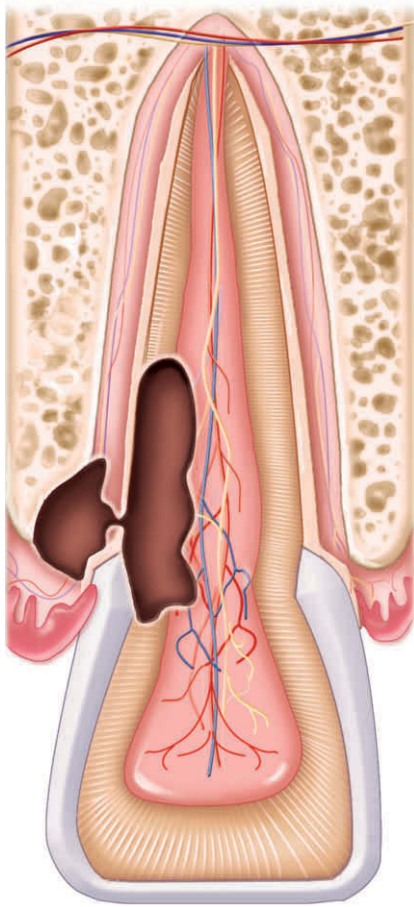
of the lesion.<sup>107</sup> Periodontal surgery can be attempted if the lesion is accessible; however, the surgery may result in unfavorable periodontal problems. If feasible, the entire lesion needs to be exposed, removed, and restored with a composite (e.g., Geristore™).

## POSTTRAUMATIC FOLLOW-UP AND MANAGEMENT

### NEED FOR ENDODONTIC TREATMENT

All avulsed teeth need to be reexamined 7 to 10 days after trauma. The splint is removed in most instances at this appointment, after the endodontic therapy is initiated, assuming the tooth has a *closed apex*. If there is an *open apex*, the tooth has the potential to revascularize, enabling the root to continue its development. Therefore these teeth should not be endodontically treated at this point, unless they show signs of infection or the tooth was out of the socket for more than 1 hour.





**Figure 6-15** CRR tends to develop in an incisal-apical direction and in most cases does not perforate through the pre-dentin or precementum until very advanced stages. The pulp is usually vital and root canal therapy has no effect on the progression of this type of resorption.

The endodontic access should be created before the removal of the splint because the tooth will be rather loose in its socket after the splint is removed. Rubber dam isolation should be used, but the traumatized tooth should not be clamped. Wedgets™ can be used instead. The canal space should be completely chemomechanically cleaned and shaped to the correct working length. Traditionally, calcium hydroxide has been the intracanal medicament of choice for traumatically injured teeth.\* It should be mixed with sterile saline or with 0.12% chlorhexidine to the consistency of toothpaste and spun into the canal with a lentulo spiral.<sup>97</sup> Other products exist that allow the clinician to inject the calcium hydroxide with a narrow disposable plastic tip; but its efficacy in filling the apical third of the canal is questionable.<sup>104</sup> Nonetheless, care must be taken not to inject the paste into the periapical tissues. As good as the calcium hydroxide is as an antibacterial agent, it has no antiinflammatory effects; it may even have the opposite effect if allowed to contact a healing PDL.<sup>22,71</sup> All indications suggest Leder-



**Figure 6-16** Radiograph showing the typical appearance of CRR: the lesion is located in the cervical area, at the level of the crestal bone.

mix Paste™ as a good alternative,<sup>29,101</sup> especially in cases in which the avulsed teeth have been exposed to extended dry times or in cases in which signs of IRR exist before the initiation of endodontic therapy.

Traditionally, an avulsed tooth was treated with an intracanal calcium hydroxide paste for several months.<sup>108</sup> It seems clear now that if calcium hydroxide is placed in an avulsed and replanted tooth within 7 to 10 days, and if there are no signs of infection or of root resorption, obturation of the canal can be completed 1 to 2 weeks later.<sup>108</sup> However, if signs of infection or IRR exist, then the thin calcium hydroxide paste that was initially placed into the canal space should be changed in about 3 weeks and a very thick (powdery) mix of calcium hydroxide and sterile saline should be packed into the root canal space. It is our recommendation not to use a radioopacifying agent, such as barium sulfate, in the calcium hydroxide mixture because adequate amounts of calcium hydroxide will appear on a radiograph with almost the same radiodensity as dentin (Fig. 6-18). If the combined calcium hydroxide/barium sulfate mixture “washes out” of the canal, the barium sulfate may remain behind, and give the appearance that the calcium hydroxide is still in the canal.<sup>4</sup> For reference purposes, it is advisable to expose a radiograph once this thick mixture has been placed in the root canal. The calcium hydroxide paste should be left in the tooth for an extended period of time until all signs of IRR have healed and until a normal PDL width has been reestablished.

\*References 22, 36, 38, 71, 97, 108, 109.



**Figure 6-17** Despite its name, CRR does not always present "cervically." Because it occurs subjacent to the epithelial attachment, the resorption may be located further apically from the cemento-enamel junction, as shown in this case.

Healing of inflammatory resorption can take anywhere from 6 to 24 months (Fig. 6-19). The calcium hydroxide filling should be examined every 3 months radiographically, and if any appears to have washed out, it should be changed.

The access of the tooth should be temporized with an appropriate restorative material. It is of utmost importance to keep all bacteria out of the canal space. Therefore, if long-term calcium hydroxide therapy is necessary, restoring the access cavity with a durable material, such as an acid etched composite, is strongly recommended.

## RECALL FREQUENCY

There are two main recall protocols for avulsed teeth. One is for roots with an open apex, and the other is for roots with a closed apex. A minimum of one periapical radiograph should be exposed at all recall appointments.

Teeth with a *fully formed apex* in which the necrotic pulp was removed within 2 weeks after trauma and then obturated a few weeks later should be recalled every 6 months for up to 5 years, and then yearly as long as possible.

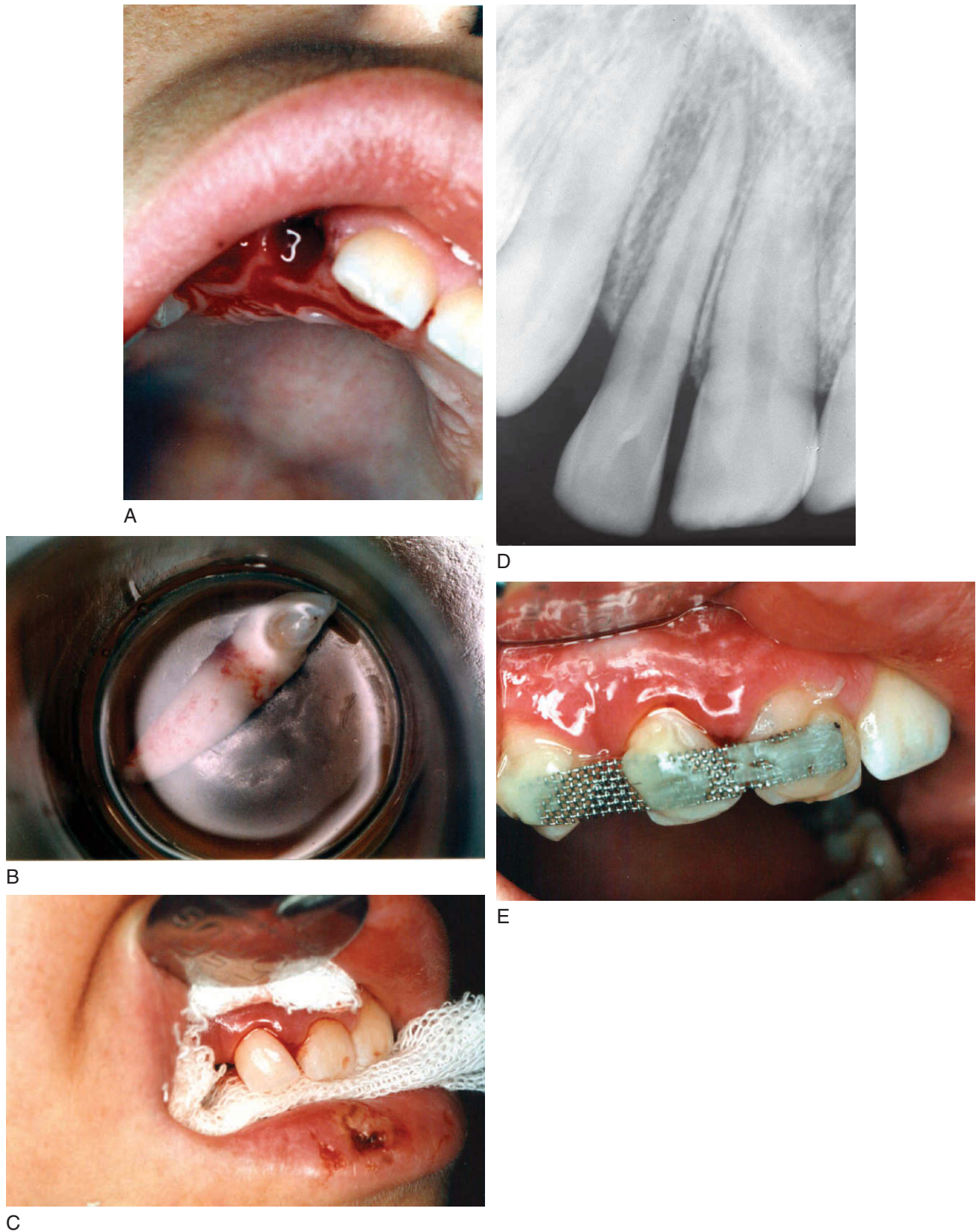
Teeth with a *fully formed apex* and in long-term calcium hydroxide treatment should be recalled every 3 months until complete resolution of the lesion is observed. After obturation, they should be recalled every 6 months.



**Figure 6-18** Placing a thick paste of calcium hydroxide (without the adjunction of radioopacifying agents such as barium sulfate) into the root canal of this maxillary right central incisor allows for evaluation of the density of the fill. In this case, the root canal space seems to be completely obliterated, indicating a good density of calcium hydroxide fill in the entire length of the root canal space.

Teeth with an *open apex* in which revascularization is expected should initially be recalled frequently or at least every 3 to 4 weeks. Once radiographic signs of further root development are observed, or if the tooth responds to sensitivity testing, the patient should be recalled every 3 to 6 months until the root is fully formed and then every 6 months for 5 years. It has been demonstrated that cold stimuli, especially carbon dioxide snow or difluorodichloromethane in an aerosol, are the best way to test pulpal sensitivity in young teeth.<sup>48</sup> The cold stimulus should be placed on the incisal edge of the tooth to prevent a false positive response from the gingivae. Alternatively, laser Doppler flowmetry has been shown to correctly predict the pulp status (vital vs. nonvital) as early as 3 weeks after replantation<sup>70,90,116</sup> (Fig. 6-20).

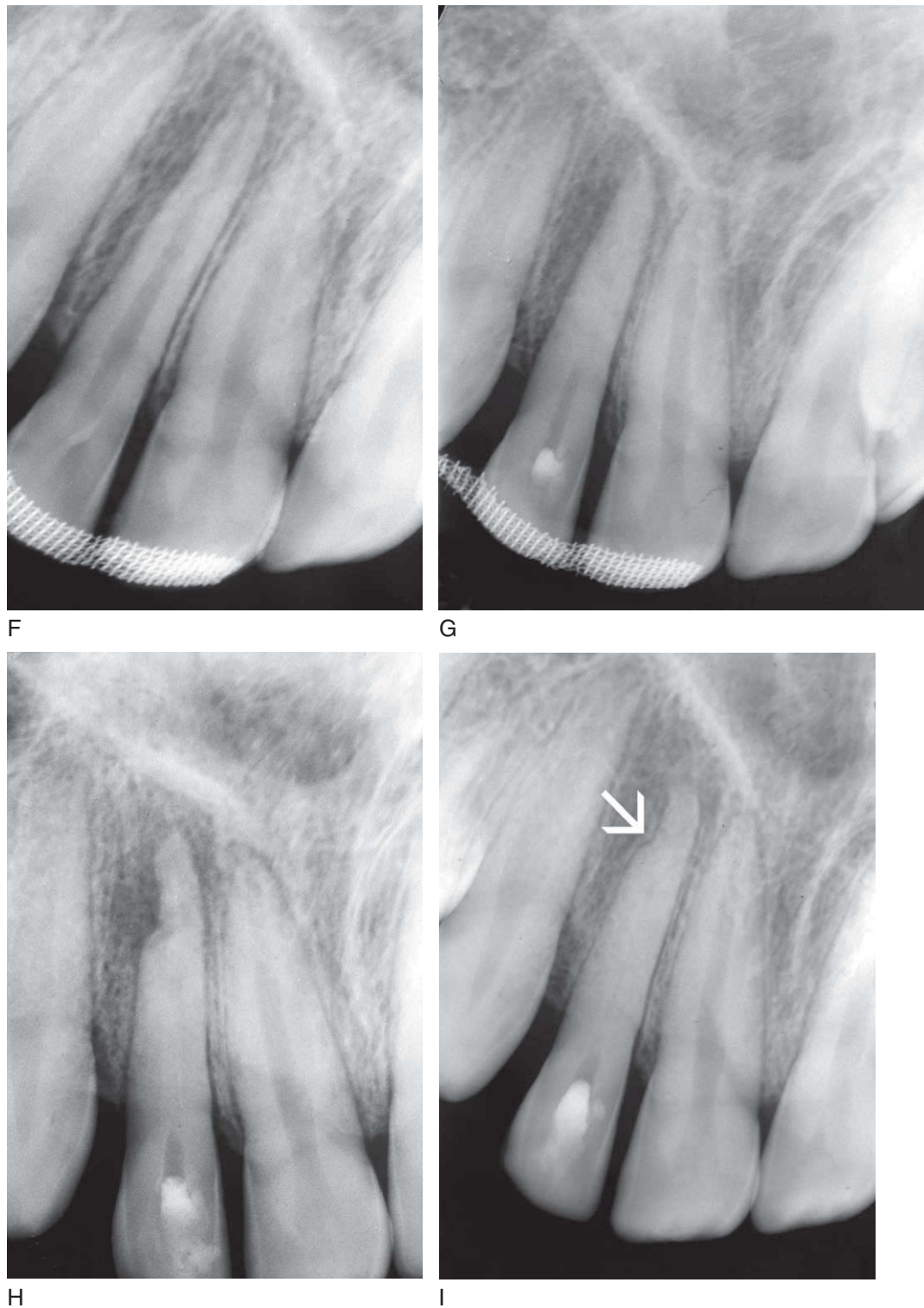
Teeth undergoing apexification treatment should be recalled frequently or at least every 3 months if they are undergoing apexification treatment (see next section). If a



**Figure 6-19** A, Observation 2 hours after avulsion, maxillary right lateral incisor. B, Storage of tooth in HBSS before replantation. C, Tooth replanted with patient biting on gauze while splint is being fabricated. D, Radiographic verification of proper repositioning of tooth. E, Tooth splinted.

*Continued*





**Figure 6-19, cont'd** **F**, Radiograph with tooth positioned in place. **G**, Radiograph with calcium hydroxide placed in canal 10 days posttrauma. **H**, Six months posttrauma, inflammatory root resorption and an associated radiolucency visible apically/distally. **I**, Twelve months posttrauma, resolution of the inflammatory root resorption and of the radiolucency are visible and the PDL has reestablished itself (*arrow*).



**Figure 6-20** When properly used, laser Doppler flowmetry will indicate as early as 2 to 3 weeks posttrauma if revascularization is occurring.

one/two step apexification procedure is performed, then they still should be recalled every 3 months for the first year and then every 6 months thereafter.

## TREATMENT OPTIONS FOR OPEN APEX TEETH THAT FAILED TO REVASCULARIZE

### Apexification

If the pulp space does not revascularize after the replantation, then it needs to undergo an apexification procedure. This should be initiated as soon as any signs of infection are evident (e.g., swelling or radiographic changes). This is very important because the remaining dentin walls are so thin that if they start to undergo IRR, a major portion of the root could be quickly lost. Also, if a root does not show any further development 3 to 6 months after replantation, then there is a good likelihood that the pulp has not revascularized; therefore an apexification procedure should be initiated before infection sets in.

An apexification is a procedure whereby the root canal of an open apex tooth is filled with calcium hydroxide to stimulate the formation of a hard tissue barrier at the apical portion of the root. After this calcified barrier has formed, it is possible to obturate the canal system by packing the obturation material without the risk of overextending the material.<sup>99</sup> There are good indications that apexification treatment not only facilitates obturation, but also allows a better outcome of involved teeth if compared with teeth in which immediate root canal obturation was performed.<sup>63</sup>

There are two steps to perform this procedure: the first is to disinfect the canal space and the second is to stimulate the apical barrier formation. Disinfection is achieved by placing a relatively thin mixture (less than toothpaste thick) of calcium hydroxide powder mixed with 0.12% chlorhexidine solution. The mixture is spun into the canal space after it has been thoroughly irrigated with 3% sodium hypochlorite solution and dried. In most cases it is not possible to thoroughly instrument the canal space because of its great width and thin walls. After 3 weeks, the patient should be recalled and the thin mixture irrigated out and dried. At this time a very thick, almost dry mixture of calcium hydroxide and sterile saline is packed to the full length of the tooth using pluggers, the blunt end of thick absorbent points, or gutta-percha cones to seat it to the full length of the root. It is usually necessary to pack the mix in several stages. Once a radiograph confirms that the mixture is dense all the way to the apex, a temporary restoration should be placed in the access cavity.

The patient should be recalled every 3 months and the density of the mixture examined radiographically. Currently, it is not recommended to replace the mixture if it appears to be intact at these recall appointments.<sup>46</sup> The time required to achieve the apical barrier formation varies between 6 and 24 months with an average of 19 months.<sup>67</sup> The barrier that forms does not have Hertwig's epithelial root sheath and has been described as osteoid and/or cementoid.<sup>51,99</sup> Once the presence of the barrier has been confirmed both radiographically and manually, the tooth can be obturated (Fig. 6-21).

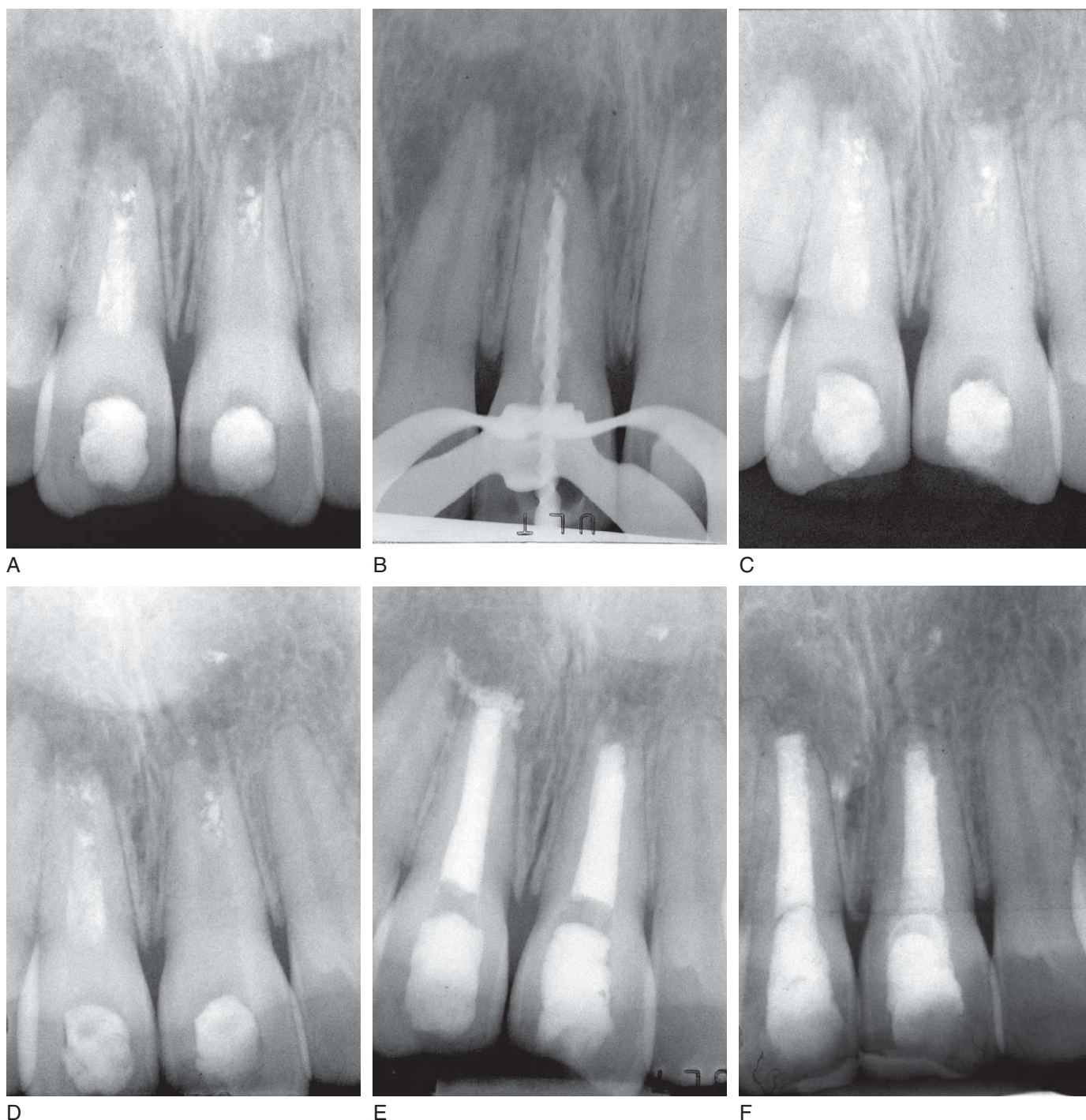
### One Versus Two Step Apexification

Recently it has been advocated to use mineral trioxide aggregate (MTA, Dentsply Tulsa Dental, Tulsa, Okla.) in a single step procedure as an apical barrier, allowing obturation in the same appointment<sup>100</sup> (see Chapter 3: Crown Fractures—Apexification Techniques). This is possible when there is no infection in the root canal system. But it is important to note that the MTA has very minimal bactericidal effect on several bacterial species.<sup>43,103</sup> Given that the presence of bacteria is the single most important factor to maintain inflammatory resorption, it is strongly advised to follow a two step apexification procedure:

1. First the canal space is disinfected for 7 to 14 days with a thin calcium hydroxide paste.
2. Next an apical barrier is created by compacting MTA (with the blunt end of a large absorbent point), followed by root canal obturation.<sup>49,102</sup>

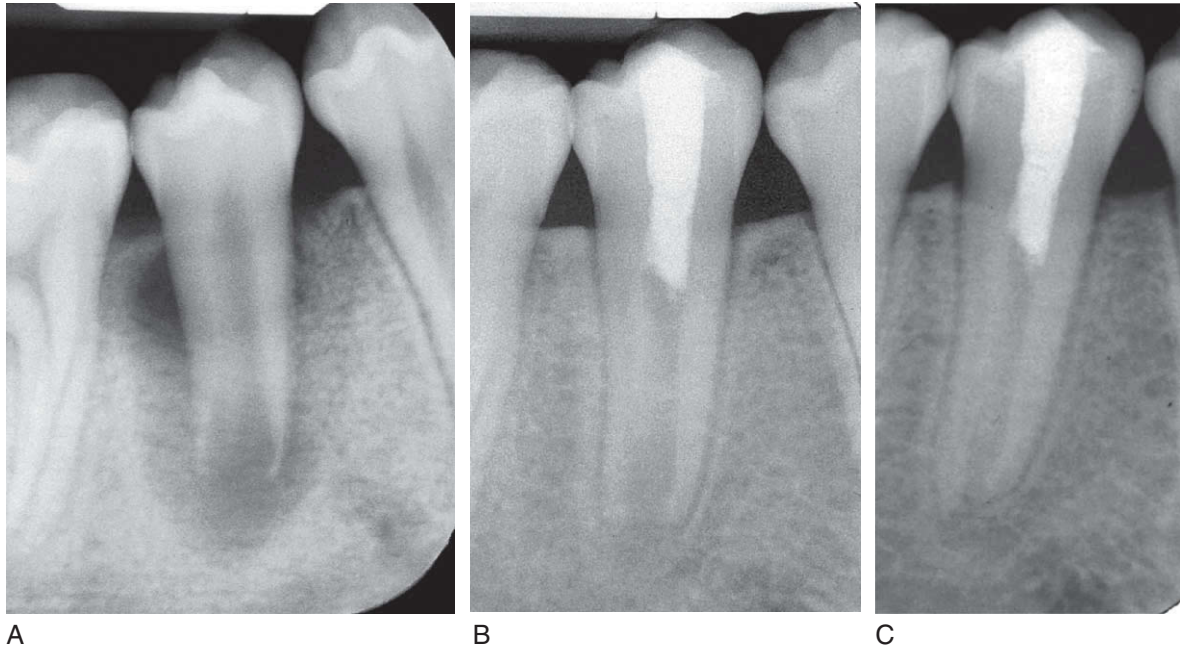
### Pulpal Regeneration Modalities

Recently, exciting case reports have been published in which the authors describe the ability to make a pulplike tissue grow inside the canal space of teeth that had an open apex with pulpal necrosis and infection<sup>26,61</sup> (Fig. 6-22). The protocol to obtain such a tissue in-growth is described in the list on p. 121.



**Figure 6-21** Both central incisors in this 13-year-old boy sustained a severe luxation injury 4 years earlier. At that time, apexification was initiated, but not followed up. **A**, Pain, facial swelling, and a radiolucent area are present periapically to both teeth. **B**, Calcium hydroxide mixed with chlorhexidine to the consistency of toothpaste was placed in the root canal to disinfect it. **C**, Four weeks later, the calcium hydroxide slurry was rinsed out and replaced by a thick mix of calcium hydroxide and water. The preexisting radiopaque deposits of barium sulphate were impossible to remove from within the root canals, even with the help of the microscope. **D**, Healing of the radiolucent periapical areas was monitored radiographically by recalling the patient every 3 months. **E**, Once an apical calcified barrier had formed, as confirmed both radiographically and clinically, the root canals were filled with a warm gutta-percha obturation technique. The apical calcified barrier is frequently porous and allows some sealer to penetrate beyond it. **F**, Two-year follow-up, with healing observed and sealer resorbed.





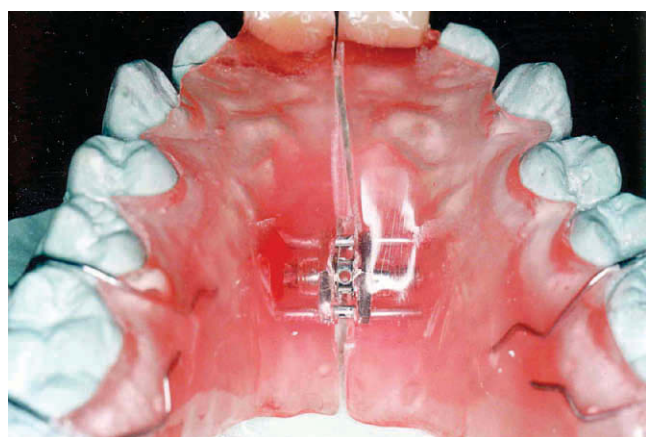
**Figure 6-22** “Regeneration” of “pulpal” tissue in infected root with open apex. **A**, Pretreatment radiograph. The pulpal space is thoroughly irrigated with sodium hypochlorite solution. Equal proportions of ciprofloxacin, metronidazole, and minocycline are mixed with sterile saline to the consistency of a paste and placed into the root canal. The mixture should be cleaned from the coronal part of the tooth because it will severely stain it. After 3 weeks, the paste is rinsed out and an endodontic instrument is introduced through the canal into the periapical tissues to create bleeding into the entire canal space. The blood is allowed to coagulate and the access cavity is closed with a bacteria-tight seal. **B**, After several weeks, the periapical area has healed and the root has continued its growth. **C**, Two-year recall shows healing and continued root growth. (Courtesy Dr. F. Banchs.)

- An access cavity is created.
- An irrigation needle is placed in the canal to within 1 mm of the apex.
- The canal is then slowly irrigated with 3% sodium hypochlorite and 0.12% chlorhexidine solutions.
- The canal is then dried with absorbent points.
- An equal mixture of ciprofloxacin, metronidazole, and minocycline paste, as described by Hoshino et al,<sup>58</sup> is prepared into a creamy consistency with sterile saline and spun into the canal with a lentulo spiral. The triple mix of antibiotics has been shown to be effective in disinfecting canal space in vivo.<sup>114</sup> Avoid leaving any of the mixture in the coronal part of the tooth because it will cause a dark stain to the dentin it contacts.
- The access cavity is temporized.
- Three weeks later the access is reopened, the canal irrigated with 3% sodium hypochlorite solution, and dried.
- A sterile instrument is placed beyond the apex to stimulate bleeding into the canal space.
- A blood clot is allowed to form in the radicular portion of the tooth by leaving the tooth open for about 15 minutes.
- The tooth is carefully closed with 3 mm of MTA compacted and desiccated with the blunt end of absorbent points on top of the blood clot, followed by a cotton pellet moistened with 0.12% chlorhexidine and temporary filling material.
- Four to six weeks later, the tooth can be permanently restored.<sup>26</sup>

## EXPECTED HEALING OUTCOME OF AVULSIONS

### EPIDEMIOLOGICAL DATA

There is now a consensus that an avulsed tooth that is treated in an appropriate and timely fashion has a good chance of survival. Average survival rate in several studies, with an observation time ranging from a few months up to 20 years,



A



B



C

**Figure 6-23** Temporary orthodontic appliance. **A**, Appliance on model to replace two central incisors. Note appliance separated at midline with palatal expansion “screw” to allow for the normal growth of the maxilla. **B**, Appliance placed in mouth. **C**, Patient with appliance in place, noting favorable esthetics.

is reported to be more than 60% (39% to 89%).<sup>7,17</sup> The most important factor affecting the survival of an avulsed tooth, apart from extraoral dry time and timely endodontic therapy, has been reported to be the stage of root development at the time of injury. A better survival rate was observed in mature teeth.<sup>17</sup> These historical studies involve statistics from treatments performed before the current knowledge about factors affecting the survival of these teeth. Therefore, it can be speculated that avulsed teeth treated with today's guidelines may have an even better chance of survival than those treated years ago.

## SIGNS OF NONHEALING

The main reason for nonhealing of an avulsed tooth in contemporary studies is ankylosis caused by extended extraoral time and/or inappropriate storage or splinting. In older studies, IRR was the main cause of nonhealing. But once it was realized that appropriate root canal therapy could

significantly reduce or eliminate it, the incidence of inflammatory resorption has notably decreased.

## TREATMENT OPTIONS

In the event that the avulsed tooth is not replanted, or should the tooth be lost because of extensive inflammatory or replacement resorption, treatment options must be considered. The primary considerations are *maintenance of alveolar bone, space maintenance, and esthetics*. Ultimately, a fixed or removable prosthesis, or an implant-supported crown, must be considered. However, typically these injuries occur in young patients when adjacent teeth have not fully erupted or when a mixed dentition is present. Since these patients are not yet good candidates for a permanent prosthesis, an interim appliance must be fabricated that allows for normal alveolar growth and eruption of the adjacent teeth.<sup>31,42,80,91,112</sup> One such device is a temporary orthodontic appliance (Fig. 6-23). This is a removable “flipper” that incorporates a



**Figure 6-24** Enamel hypoplasia of the maxillary left central incisor secondary to reimplanting an avulsed primary central incisor.

pontic(s) on an orthodontic retainer. The appliance is separated at the midline, with a palatal expansion “screw” between the halves. This allows the appliance to be adjusted over time to permit normal maxillary growth. Depending on the period of time until a fixed prosthesis can be placed, the appliance may need replacement periodically to allow for the continued development of the dentition.

## AVULSED PRIMARY TEETH

Avulsed primary teeth should not be replanted. This is because of the very high risk of damaging the forming permanent tooth (Fig. 6-24), either by the replantation procedure itself or secondarily if the necrotic pulp of the replanted primary tooth becomes infected.<sup>32,66,118</sup>

## CONCLUSION

It is clear that an avulsed tooth can be predictably saved if treated in a proper and timely fashion and if every effort is made to prevent any further injury to the attached PDL.

- An avulsed tooth should be replanted as soon as possible, preferably at the location of the injury.
- If it is impossible to replant the avulsed tooth immediately, then it needs to be placed in a proper storage medium like HBSS, milk, or physiological saline.
- Once the tooth is replanted, it should be splinted for 7 to 10 days with a semiflexible physiological splint.
- Pulp revascularization should be attempted on avulsed teeth with an open apex.

- Endodontic therapy should be initiated after 7 to 10 days if the avulsed tooth has a closed apex.
- Endodontic treatment of avulsed teeth should include the use of an intracanal medication to disinfect the root canal system.
- All avulsed teeth should be carefully monitored initially, after a few weeks, and then at least every 6 months during the next 5 years.

## REFERENCES

1. Abbott PV: Medicaments: aids to success in endodontics. Part 1. A review of the literature, *Aust Dent J* 35:438-448, 1990.
2. Abbott PV: Systemic release of corticosteroids following intra-dental use, *Int Endod J* 25:189-191, 1992.
3. Abbott PV, Heithersay GS, Hume WR: Release and diffusion through human tooth roots in vitro of corticosteroid and tetracycline trace molecules from Ledermix Paste, *Endod Dent Traumatol* 4:55-62, 1988.
4. Alacam T, Gorgul G, Omurlu H: Evaluation of diagnostic radiopaque contrast materials used with calcium hydroxide, *J Endod* 16:365-368, 1990.
5. Andersson L, Blomlof L, Lindsog S et al: Tooth ankylosis. Clinical, radiographic and histological assessments, *Int J Oral Surg* 13:423-431, 1984.
6. Andersson L, Jonsson BG, Hammarstrom L et al: Evaluation of statistics and desirable experimental design of a histomorphometrical method for studies of root resorption, *Endod Dent Traumatol* 3:288-295, 1987.
7. Andreasen FM, Andreasen JO: Avulsion. In Andreasen JO, Andreasen FM, editors: *Textbook and color atlas of traumatic injuries to the teeth*, Copenhagen, 1994, Munksgaard Intl.
8. Andreasen JO: Etiology and pathogenesis of traumatic dental injuries. A clinical study of 1298 cases, *Scand J Dent Res* 78:339-342, 1970.
9. Andreasen JO: Analysis of pathogenesis and topography of replacement root resorption (ankylosis) after replantation of mature permanent incisors in monkeys, *Swed Dent J* 4:231-240, 1980.
10. Andreasen JO: Analysis of topography of surface and inflammatory root resorption after replantation of mature permanent incisors in monkeys, *Swed Dent J* 4:135-144, 1980.
11. Andreasen JO: The effect of removal of the coagulum in the alveolus before replantation upon periodontal and pulpal healing of mature permanent incisors in monkeys, *Int J Oral Surg* 9:458-461, 1980.
12. Andreasen JO: A time-related study of periodontal healing and root resorption activity after replantation of mature permanent incisors in monkeys, *Swed Dent J* 4:101-110, 1980.
13. Andreasen JO: Effect of extraalveolar period and storage media upon periodontal and pulpal healing after replantation of mature permanent incisors in monkeys, *Int J Oral Surg* 10:43-53, 1981.
14. Andreasen JO: The effect of pulp extirpation or root canal treatment on periodontal healing after replantation of permanent incisors in monkeys, *J Endod* 7:245-252, 1981.
15. Andreasen JO: Relationship between surface and inflammatory resorption and changes in the pulp after replantation of permanent incisors in monkeys, *J Endod* 7:294-301, 1981.



16. Andreasen JO, Borum MK, Andreasen FM: Replantation of 400 avulsed permanent incisors. 3. Factors related to root growth, *Endod Dent Traumatol* 11:69-75, 1995.
17. Andreasen JO, Borum MK, Jacobsen HL et al: Replantation of 400 avulsed permanent incisors. 1. Diagnosis of healing complications, *Endod Dent Traumatol* 11:51-58, 1995.
18. Andreasen JO, Borum MK, Jacobsen HL et al: Replantation of 400 avulsed permanent incisors. 2. Factors related to pulpal healing, *Endod Dent Traumatol* 11:59-68, 1995.
19. Andreasen JO, Borum MK, Jacobsen HL et al: Replantation of 400 avulsed permanent incisors. 4. Factors related to periodontal ligament healing, *Endod Dent Traumatol* 11:76-89, 1995.
20. Andreasen JO, Hjorting-Hansen E: Replantation of teeth. I. Radiographic and clinical study of 110 human teeth replanted after accidental loss, *Acta Odontol Scand* 24:263-286, 1966.
21. Andreasen JO, Hjorting-Hansen E: Replantation of teeth. II. Histological study of 22 replanted anterior teeth in humans, *Acta Odontol Scand* 24:287-306, 1966.
22. Andreasen JO, Kristerson L: The effect of extraalveolar root filling with calcium hydroxide on periodontal healing after replantation of permanent incisors in monkeys, *J Endod* 7:349-354, 1981.
23. Andreasen JO, Kristerson L: Evaluation of different types of autotransplanted connective tissues as potential periodontal ligament substitutes. An experimental replantation study in monkeys, *Int J Oral Surg* 10:189-201, 1981.
24. Andreasen JO, Kristerson L: The effect of limited drying or removal of the periodontal ligament. Periodontal healing after replantation of mature permanent incisors in monkeys, *Acta Odontol Scand* 39:1-13, 1981.
25. Andreasen JO, Reinholdt J, Riis I et al: Periodontal and pulpal healing of monkey incisors preserved in tissue culture before replantation, *Int J Oral Surg* 7:104-112, 1978.
26. Banchs F, Trope M: Revascularization of immature permanent teeth with apical periodontitis: new treatment protocol? *J Endod* 30:196-200, 2004.
27. Bjorvatn K, Selvig KA, Klinge B: Effect of tetracycline and SnF<sub>2</sub> on root resorption in replanted incisors in dogs, *Scand J Dent Res* 97:477-482, 1989.
28. Blomlof L: Milk and saliva as possible storage media for traumatically exarticulated teeth prior to replantation, *Swed Dent J Suppl* 8:1-26, 1981.
29. Bryson EC, Levin L, Banchs F et al: Effect of immediate intracanal placement of Ledermix Paste(R) on healing of replanted dog teeth after extended dry times, *Dent Traumatol* 18:316-321, 2002.
30. Campbell KM, Casas MJ, Kenny DJ et al: Diagnosis of ankylosis in permanent incisors by expert ratings. Periotest and digital sound wave analysis, *Dent Traumatol* 21:206-212, 2005.
31. Choonara SA: Orthodontic space maintenance—a review of current concepts and methods *SADJ* 60:113, 115-117, 2005.
32. Christophersen P, Freund M, Harild L: Avulsion of primary teeth and sequelae on the permanent successors, *Dent Traumatol* 21:320-323, 2005.
33. Cohen S, Blanco L, Berman LH: Early radiographic diagnosis of inflammatory root resorption, *Gen Dent* 51:235-240, 2003.
34. Crona-Larsson G, Noren JG: Luxation injuries to permanent teeth—a retrospective study of etiological factors, *Endod Dent Traumatol* 5:176-179, 1989.
35. Cummings GR, Torabinejad M: Effect of systemic doxycycline on alveolar bone loss after periradicular surgery, *J Endod* 26:325-327, 2000.
36. Cvek M: Treatment of non-vital permanent incisors with calcium hydroxide. I. Follow-up of periapical repair and apical closure of immature roots, *Odontol Revy* 23:27-44, 1972.
37. Cvek M: Clinical procedures promoting apical closure and arrest of external root resorption in non-vital permanent incisors, *Trans Int Conf Endod* 5:30-41, 1973.
38. Cvek M: Treatment of non-vital permanent incisors with calcium hydroxide. II. Effect on external root resorption in luxated teeth compared with effect of root filling with gutta-percha. A follow-up, *Odontol Revy* 24:343-354, 1973.
39. Cvek M, Cleaton-Jones P, Austin J et al: Effect of topical application of doxycycline on pulp revascularization and periodontal healing in reimplanted monkey incisors, *Endod Dent Traumatol* 6:170-176, 1990.
40. Cvek M, Cleaton-Jones P, Austin J et al: Pulp revascularization in reimplanted immature monkey incisors—predictability and the effect of antibiotic systemic prophylaxis, *Endod Dent Traumatol* 6:157-169, 1990.
41. Doyle DL, Dumsha TC, Sydiskis RJ: Effect of soaking in Hank's balanced salt solution or milk on PDL cell viability of dry stored human teeth, *Endod Dent Traumatol* 14:221-224, 1998.
42. Duward CS: Space maintenance in the primary and mixed dentition, *Ann R Australas Coll Dent Surg* 15:203-205, 2000.
43. Estrela C, Bammann LL, Estrela CR et al: Antimicrobial and chemical study of MTA, Portland cement, calcium hydroxide paste, Sealapex and Dycal, *Braz Dent J* 11:3-9, 2000.
44. Filippi A, Pohl Y, von Arx T: Treatment of replacement resorption with Emdogaim—preliminary results after 10 months, *Dent Traumatol* 17:134-138, 2001.
45. Filippi A, Pohl Y, von Arx T: Treatment of replacement resorption with Emdogain—a prospective clinical study, *Dent Traumatol* 18:138-143, 2002.
46. Flores MT, Andreasen JO, Bakland LK et al: Guidelines for the evaluation and management of traumatic dental injuries, *Dent Traumatol* 17:193-198, 2001.
47. Forti G, Benincori C: Doxycycline and the teeth, *Lancet* 1:782, 1969.
48. Fuss Z, Trowbridge H, Bender IB et al: Assessment of reliability of electrical and thermal pulp testing agents, *J Endod* 12:301-305, 1986.
49. Giuliani V, Baccetti T, Pace R: The use of MTA in teeth with necrotic pulps and open apices, *Dent Traumatol* 18:217-221, 2002.
50. Grevstad HJ: Doxycycline prevents root resorption and alveolar bone loss in rats after periodontal surgery, *Scand J Dent Res* 101:287-291, 1993.
51. Ham JW, Patterson SS, Mitchell DF: Induced apical closure of immature pulpless teeth in monkeys, *Oral Surg Oral Med Oral Pathol* 33:438-449, 1972.
52. Hammarstrom L, Blomlof L, Feiglin B et al: Replantation of teeth and antibiotic treatment, *Endod Dent Traumatol* 2:51-57, 1986.
53. Hammarstrom L, Lindsbog S: Factors regulating and modifying dental root resorption, *Proc Finn Dent Soc* 88 Suppl 1:115-123, 1992.
54. Hammarstrom L, Lindsbog S: General morphological aspects of resorption of teeth and alveolar bone, *Int Endod J* 18:93-108, 1985.
55. Harrington GW, Natkin E: External resorption associated with bleaching of pulpless teeth, *J Endod* 5:344-348, 1979.
56. Heimdahl A, Nord CE: Antimicrobial agents in the treatment of periodontal diseases: special aspects on tetracycline and doxycycline, *Scand J Infect Dis Suppl* 53:35-45, 1988.
57. Hiltz J, Trope M: Vitality of human lip fibroblasts in milk, Hanks balanced salt solution and Viaspan storage media, *Endod Dent Traumatol* 7:69-72, 1999.
58. Hoshino E, Kurihara-Ando N, Sato I et al: In-vitro antibacterial susceptibility of bacteria taken from infected root

- dentine to a mixture of ciprofloxacin, metronidazole and minocycline, *Int Endod J* 29:125-130, 1996.
59. Hupp JG, Mesaros SV, Aukhil I et al: Periodontal ligament vitality and histologic healing of teeth stored for extended periods before transplantation, *Endod Dent Traumatol* 14:79-83, 1998.
  60. Iqbal MK, Bamaas N: Effect of enamel matrix derivative (EMDOGAIN) upon periodontal healing after replantation of permanent incisors in beagle dogs, *Dent Traumatol* 17:36-45, 2001.
  61. Iwaya SI, Ikawa M, Kubota M: Revascularization of an immature permanent tooth with apical periodontitis and sinus tract, *Dent Traumatol* 17:185-187, 2001.
  62. Jarvinen S: Fractured and avulsed permanent incisors in Finnish children. A retrospective study, *Acta Odontol Scand* 37:47-50, 1979.
  63. Kerekes K, Heide S, Jacobsen I: Follow-up examination of endodontic treatment in traumatized juvenile incisors, *J Endod* 6:744-748, 1980.
  64. Kim ST, Abbott PV, McGinley P: The effects of Ledermix Paste on discoloration of immature teeth, *Int Endod J* 33:233-237, 2000.
  65. Kim ST, Abbott PV, McGinley P: The effects of Ledermix Paste on discoloration of mature teeth, *Int Endod J* 33:227-232, 2000.
  66. Kinoshita S, Mitomi T, Taguchi Y et al: Prognosis of replanted primary incisors after injuries, *Endod Dent Traumatol* 16:175-183, 2000.
  67. Kleier DJ, Barr ES: A study of endodontically apexified teeth, *Endod Dent Traumatol* 7:112-117, 1991.
  68. Kling M, Cvek M, Mejare I: Rate and predictability of pulp revascularization in therapeutically reimplanted permanent incisors, *Endod Dent Traumatol* 2:83-89, 1986.
  69. Lam K, Sae-Lim V: The effect of Emdogain gel on periodontal healing in replanted monkeys' teeth, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 97:100-107, 2004.
  70. Lee JY, Yanpiset K, Sigurdsson A et al: Laser Doppler flowmetry for monitoring traumatized teeth, *Dent Traumatol* 17:231-235, 2001.
  71. Lengheden A, Blomlof L, Lindsog S: Effect of immediate calcium hydroxide treatment and permanent root-filling on periodontal healing in contaminated replanted teeth, *Scand J Dent Res* 99:139-146, 1991.
  72. Lenstrup K, Skieller V: A follow-up study of teeth replanted after accidental loss, *Acta Odontol Scand* 17:503-509, 1959.
  73. Lindsog S, Blomlof L: Influence of osmolality and composition of some storage media on human periodontal ligament cells, *Acta Odontol Scand* 40:435, 1982.
  74. Lindsog S, Blomlof L, Hammarstrom L: Repair of periodontal tissues in vivo and in vitro, *J Clin Periodontol* 10:188-205, 1983.
  75. Malmgren B, Cvek M, Lundberg M et al: Surgical treatment of ankylosed and infrapositioned reimplanted incisors in adolescents, *Scand J Dent Res* 92:391-399, 1984.
  76. Malmgren B, Malmgren O: Rate of infra-position of reimplanted ankylosis incisors related to age and growth in children and adolescents, *Dental Traumatol* 18:28-36, 2002.
  77. Mandel U, Viidik A: Effect of splinting on the mechanical and histological properties of the healing periodontal ligament in the vervet monkey (*Cercopithecus aethiops*), *Arch Oral Biol* 34:209-217, 1989.
  78. Martin MP, Pileggi R: A quantitative analysis of Propolis: a promising new storage media following avulsion, *Dent Traumatol* 20:85-89, 2004.
  79. Mavragani M, Brudvik P, Selvig KA: Orthodontically induced root and alveolar bone resorption: inhibitory effect of systemic doxycycline administration in rats, *Eur J Orthod* 27:215-225, 2005.
  80. McKeown HF, Sandler J: The two by four appliance: a versatile appliance, *Dent Update* 28:496-500, 2001.
  81. Nasjleti CE, Castelli WA, Caffesse RG: The effects of different splinting times on replantation of teeth in monkeys, *Oral Surg Oral Med Oral Pathol* 53:557-566, 1982.
  82. Ng VW, Bissada NF: Clinical evaluation of systemic doxycycline and ibuprofen administration as an adjunctive treatment for adult periodontitis, *J Periodontol* 69:772-776, 1998.
  83. O'Brien TP, Roszkowski MT, Wolff LF et al: Effect of a non-steroidal anti-inflammatory drug on tissue levels of immunoreactive prostaglandin E<sub>2</sub>, immunoreactive leukotriene, and pain after periodontal surgery, *J Periodontol* 67:1307-1316, 1996.
  84. Öhman A: Healing and sensitivity to pain in young replanted human teeth. An experimental, clinical and histological study, *Odontol Tidskr* 73:168-227, 1965.
  85. Oikarinen K, Kassila O: Causes and types of traumatic tooth injuries treated in a public dental health clinic, *Endod Dent Traumatol* 3:172-177, 1987.
  86. Oswald RJ, Harrington GW, Van Hassel HJ: Replantation 1. The role of the socket, *J Endod* 6:479-484, 1980.
  87. Pettiette M, Hupp J, Mesaros S et al: Periodontal healing of extracted dogs' teeth air-dried for extended periods and soaked in various media, *Endod Dent Traumatol* 13:113-118, 1997.
  88. Pierce A, Heithersay G, Lindsog S: Evidence for direct inhibition of dentinoclasts by a corticosteroid/antibiotic endodontic paste, *Endod Dent Traumatol* 4:44-45, 1988.
  89. Pohl Y, Tekin U, Boll M et al: Investigations on a cell culture medium for storage and transportation of avulsed teeth, *Aust Endod J* 25:70-75, 1999.
  90. Ritter AL, Ritter AV, Murrah V et al: Pulp revascularization of replanted immature dog teeth after treatment with minocycline and doxycycline assessed by laser Doppler flowmetry, radiography, and histology, *Dent Traumatol* 20:75-84, 2004.
  91. Robertson S, Mohlin B: The congenitally missing upper lateral incisor: a retrospective study of orthodontic space closure versus restorative treatment, *Eur J Orthod* 22:697-710, 2000.
  92. Sae-Lim V, Wang CY, Trope M: Effect of systemic tetracycline and amoxicillin on inflammatory root resorption of replanted dogs' teeth, *Endod Dent Traumatol* 14:216-220, 1998.
  93. Saxen L, Asikainen S, Kanervo A et al: The long-term efficacy of systemic doxycycline medication in the treatment of localized juvenile periodontitis, *Arch Oral Biol* 35 Suppl:227S-229S, 1990.
  94. Schjott M, Andreasen JO: Emdogain does not prevent progressive root resorption after replantation of avulsed teeth: a clinical study, *Dent Traumatol* 21:46-50, 2005.
  95. Skoglund A, Tronstad L: Pulpal changes in replanted and autotransplanted immature teeth of dogs, *J Endod* 7:309-316, 1981.
  96. Sigalas E, Regan JD, Kramer PR et al: Survival of human periodontal ligament cells in media proposed for transport of avulsed teeth, *Dent Traumatol* 20:21-28, 2004.
  97. Sigurdsson A, Stancill R, Madison S: Intracanal placement of Ca(OH)<sub>2</sub>: a comparison of techniques, *J Endod* 18:367-370, 1992.
  98. Solder PO, Otteskog P, Andreasen JO et al: Effect of drying on viability of periodontal membrane, *Scand J Dent Res* 85:164-168, 1977.
  99. Steiner JC, Dow PR, Cathey GM: Inducing root end closure of nonvital permanent teeth, *J Dent Child* 35:47-54, 1968.
  100. Steinig TH, Regan JD, Gutmann JL: The use and predictable placement of mineral trioxide aggregate in one-visit apexification cases, *Aust Endod J* 29:34-42, 2003.

101. Thong YL, Messer HH, Siar CH et al: Periodontal response to two intracanal medicaments in replanted monkey incisors, *Dent Traumatol* 17:254-259, 2001.
102. Torabinejad M, Chivian N: Clinical applications of mineral trioxide aggregate, *J Endod* 25:197-205, 1999.
103. Torabinejad M, Hong CU, Pitt Ford TR et al: Antibacterial effects of some root end filling materials, *J Endod* 21:403-406, 1995.
104. Torres CP, Apicella M, Yancich P et al: Intracanal placement of calcium hydroxide: a comparison of techniques, revisited, *J Endod* 30:225-227, 2004.
105. Tronstad L: Root resorption—etiology, terminology and clinical manifestations, *Endod Dent Traumatol* 4:241-252, 1988.
106. Trope M: Cervical root resorption, *J Am Dent Assoc* 128 Suppl:56S-59S, 1997.
107. Trope M: Root resorption of dental and traumatic origin: classification based on etiology, *Pract Periodontics Aesthet Dent* 10:515-522, 1998.
108. Trope M, Moshonov J, Nissan R et al: Short vs. long-term calcium hydroxide treatment of established inflammatory root resorption in replanted dog teeth, *Endod Dent Traumatol* 11:124-128, 1995.
109. Trope M, Yesilsoy C, Koren L et al: Effect of different endodontic treatment protocols on periodontal repair and root resorption of replanted dog teeth, *J Endod* 18:492-496, 1992.
110. Troullos E, Hargreaves KM, Dionne RA: Ibuprofen elevates immunoreactive beta-endorphin levels in humans during surgical stress, *Clin Pharmacol Ther* 62:74-81, 1997.
111. Troullos ES, Hargreaves KM, Butler DP et al: Comparison of nonsteroidal anti-inflammatory drugs, ibuprofen and flurbiprofen, with methylprednisolone and placebo for acute pain, swelling, and trismus, *J Oral Maxillofac Surg* 48:945-952, 1990.
112. Ward S, Read MJ: The contemporary use of removable orthodontic appliances, *Dent Update* 31:215-218, 2004.
113. Williams RC, Jeffcoat MK, Howell TH et al: Ibuprofen: an inhibitor of alveolar bone resorption in beagles, *J Periodontal Res* 23:225-229, 1988.
114. Windley W III, Teixeira F, Levin L et al: Disinfection of immature teeth with a triple antibiotic paste, *J Endod* 31:439-443, 2005.
115. Van Hassel HJ, Oswald RJ, Harrington GW: Replantation 2. The role of the periodontal ligament, *J Endod* 6:506-508, 1980.
116. Yanpiset K, Vongsavan N, Sigurdsson A et al: Efficacy of laser Doppler flowmetry for the diagnosis of revascularization of reimplanted immature dog teeth, *Dent Traumatol* 17:63-70, 2001.
117. Zamet JS: A comparative clinical study of three periodontal surgical techniques, *J Clin Periodontol* 2:87-97, 1975.
118. Zamon E L, Kenny DJ: Replantation of avulsed primary incisors: a risk-benefit assessment, *J Can Dent Assoc* 67:386, 2001.



# ALVEOLAR FRACTURES



RADHIKA CHIGURUPATI AND KENNETH H. DAWSON

## CHAPTER OUTLINE

### EPIDEMIOLOGY AND ETIOLOGY

### TERMINOLOGY AND CLASSIFICATION

### CLINICAL AND RADIOGRAPHIC FINDINGS

### TREATMENT

#### Antibiotic Treatment and Tetanus Prophylaxis

#### Reduction

#### Fixation and Immobilization

##### Composite Retained Wire Splint

##### Arch Bars

##### Acrylic Splint

##### Orthodontic Appliances

##### Plate-and-Screw Fixation

#### Rehabilitation

### HEALING

#### Sequence of Fracture Healing

### COMPLICATIONS

#### Malocclusion

#### Loss of Alveolar Bone

#### Loss of Teeth

### ENDODONTIC IMPLICATIONS

### PROGNOSIS AND OUTCOME ASSESSMENT

### PEDIATRIC CONSIDERATIONS

### CONCLUSION

Injuries to the teeth and the supporting alveolar bone may often go undiagnosed in the hospital emergency room because of the urgency of other associated critical injuries or the lack of adequate training to evaluate these injuries. Displaced alveolar fractures and associated dental injuries are to be considered a subacute emergency because the timing of treatment affects the prognosis of the involved teeth.<sup>8</sup> Whenever possible, these injuries should be treated without delay to improve the prognosis of the teeth, reduce pain, and restore function.

Alveolar fractures often occur with concomitant dental and soft tissue injuries and sometimes with facial bone fractures. These fractures result in injury not only to the supporting bone, but also to the pulps and periodontal ligaments of the involved teeth. Treatment of these injuries requires immediate reduction, fixation and immobilization, and long-term follow-up and restoration of the injured teeth. Every effort should be made to preserve alveolar bone and restorable teeth. The mucogingival tissue must be properly reapproximated to prevent an unesthetic deformity that may be difficult to reconstruct at a later date. An endodontic assessment of all injured teeth should be planned along with the prosthetic rehabilitation of the fractured or avulsed teeth. Pulpal necrosis, root resorption, loss of bone and teeth, and malocclusion are some of the complications of alveolar fractures.

This chapter will discuss the terminology, recognition, evaluation, management, and endodontic implications of alveolar fractures with examples of some clinical scenarios. Awareness of these basic principles will help the dentist to provide timely care and create an understanding of when to promptly refer these injuries.

## EPIDEMIOLOGY AND ETIOLOGY

Alveolar fractures are usually reported in conjunction with dental injuries and facial fractures, making it difficult to estimate their incidence. In an investigation of patients coming

to the University Dental Hospital at Copenhagen,<sup>6,41</sup> alveolar fractures accounted for 9% of all dento-alveolar injuries to the permanent dentition. Another study<sup>34</sup> at a medical center in Iran retrospectively reviewed maxillo-facial injuries in 237 patients over 5 years and found that alveolar fractures accounted for approximately 2% of all mandibular and 9% of all maxillary fractures. Reviewing 174 patients younger than 16 years of age, a study at the Osaka University Dental Hospital<sup>25</sup> revealed that mandibular fractures (56%) were the most common form of maxillofacial injury, with dento-alveolar fractures being the second most common, accounting for 31% of all maxillofacial injuries.

Alveolar fractures can occur in any age group, but are most commonly seen in the first and second decade after eruption of permanent incisors.<sup>6,19,25</sup> A review of 9543 cases of cranio-maxillo-facial trauma reported to the University Hospital of Innsbruck<sup>22</sup> revealed that the mean age of all dento-alveolar injuries was 18 years, with a standard deviation of 15 years. Most epidemiological studies have shown that males are twice as likely as females to sustain dento-alveolar and facial trauma for all age groups.<sup>2,6,22,25</sup>

The cause of alveolar fractures varies with different age groups. Among *children*, alveolar fractures are often caused by bicycle injuries, falls, sports injuries, and occasionally by motor vehicular crashes and child abuse.<sup>2,14,22,25,35</sup> For *adults*, alveolar fractures are often caused by fighting, motor vehicle crashes, falls, contact sports injuries, and occasionally, industrial trauma.<sup>2,23,31,44</sup>

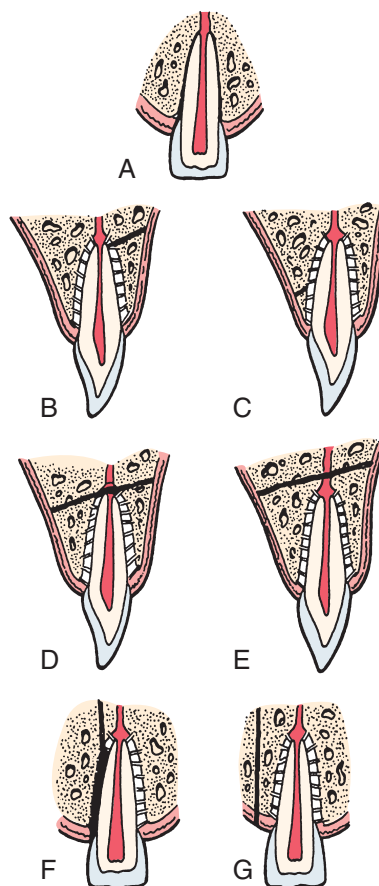
Alveolar fractures occur predominantly in dentate arches, but they can also occur in edentulous arches. The anterior maxilla and mandible are the most common sites for alveolar fractures because of the location and vulnerability of these anterior regions.<sup>14,23,31,33,44</sup> Increased maxillary incisor overjet and proclination, and incompetent lips are other factors that predispose the anterior maxilla to dento-alveolar injuries.<sup>26</sup> The anatomical shape and high density of buccal and lingual cortical bone make the posterior mandible more resistant to alveolar fractures.<sup>33</sup> The maxillary alveolus and tuberosity can also occasionally fracture secondary to the extraction of a tooth.<sup>12</sup>

fractures, the periosteum and associated periodontal ligament (PDL) are usually severely injured and there may be an intrusion or fracture of the involved teeth.<sup>12,44</sup>

Andreassen<sup>7</sup> classified injuries of the supporting bone into the following types:

1. **Comminution of the alveolar socket:** crushing of the alveolar socket associated with an intrusive or lateral luxation of an associated tooth or teeth.
2. **Fracture of the alveolar socket wall:** the fracture is usually confined to the facial or lingual cortical bone.
3. **Fracture of the alveolar process:** the fracture involves both labial and lingual cortical bone, which may or may not involve the tooth socket.
4. **Fracture of the mandible or maxilla:** the fracture involves the base of the mandible or maxilla and the alveolar process. This fracture may or may not involve the alveolar socket.

Figure 7-1 further illustrates injuries to the supporting bone.<sup>40</sup>



**Figure 7-1** Injuries to the supporting bone. **A**, Comminution of the alveolar socket. **B** and **C**, Fracture of the alveolar socket wall. **D** and **E**, Fracture of the alveolar process. **F** and **G**, Fracture of the mandible and maxilla. (Modified from Powers MP, Quereshy FA, Ramsey CA: Diagnosis and management of dentoalveolar injuries. In Fonseca JR et al, editors: Oral and maxillofacial trauma, vol 1, ed 3, St Louis, 2005, Saunders.)

## TERMINOLOGY AND CLASSIFICATION

Alveolar fractures are typically referred to as *dento-alveolar fractures* because of the involvement of associated teeth. As defined below, these fractures are usually compound (open) fractures and occasionally may be *comminuted*.<sup>24</sup> *Open* fractures involve the tooth bearing area of the maxilla or mandible, whereby the fracture has as an extraoral or intra-oral wound. *Comminution* refers to shattering of the bone into small fragments and usually occurs as a result of a high energy impact over a limited area. In comminuted alveolar

## CLINICAL AND RADIOGRAPHIC FINDINGS

Although alveolar fractures may only involve an isolated tooth, they usually involve two or more teeth with labial or lingual displacement of a dento-alveolar segment, resulting in the loss of arch continuity and painful occlusal interferences. In the maxillary arch, typically the dento-alveolar segment is displaced in a palatal and inferior direction, with the fracture occurring on the labial or buccal alveolar socket wall. In the mandible, especially in the anterior region, both labial and lingual alveolar socket wall fractures usually occur, with the displacement of the segment directed labially or lingually<sup>33</sup>; there may be associated luxations, avulsions, or fractures of the teeth within the segment.<sup>7</sup>

Upon palpation, there is tenderness and discomfort with mobility of the entire bony segment; sometimes there is *crepitation* (a crackling or popping sound) as the fractured segment is moved. Percussion of the teeth within the fractured alveolar segment may generate a dull note, which has been referred to as the sound of a “cracked pot.”<sup>12,33,44</sup> Occasionally, there may be extensive comminution, which may result in the loss of alveolar bone and teeth. Fractures of the edentulous alveolar process are not very common. But when they occur, the overlying mucosa may or may not be intact, with the possibility of developing an oronasal or oroantral communication.<sup>44</sup> Soft tissue injuries accompanying alveolar fractures often present as contusion of mucosal tissues, tear of muco-gingival tissues, or laceration of the lip (Fig. 7-2).

Initial treatment may sometimes need to be performed without radiographic examination to avoid delay. For an isolated dento-alveolar fracture, periapical and panoramic radiographs are strongly recommended (when possible) before treatment. Periapical and panoramic radiographs may reveal horizontal fracture lines, usually above the apices or at the level of the apices of the teeth. Vertical fracture lines are usually seen along the PDL space of the teeth or in the interdental bone, and have a lateral or apical widening of the PDL space, indicating a luxation or displacement of the associated teeth.<sup>5,40</sup> Periapical radiographs are also very useful in monitoring the traumatized teeth over time (Fig. 7-3). Occlusal and true lateral view radiographs are useful in identifying and confirming the fracture of the alveolar socket wall and the direction of the displaced tooth/teeth. Lateral views should include the soft tissue of the lip because they are useful in detecting the presence of soft tissue embedded radiopaque foreign bodies or tooth fragments. In injuries of the primary dentition, these lateral views help to determine the position of displaced primary teeth and their relationship to the developing permanent tooth bud and alveolar socket wall<sup>10</sup> (Fig. 7-4). Computerized tomographic (CT) scans of the face are often used to diagnose facial fractures and head injuries. Maxillary fractures involving the alveolar process and palate may be more apparent on CT scans as opposed to conventional radiographs (Fig. 7-5).



A



B



C

**Figure 7-2** A, Laceration of lower lip accompanied by dento-alveolar fractures in a 5-year-old child involved in a motor vehicle crash. B, Lip laceration débrided and cleansed. C, Repair of laceration completed after treatment of dento-alveolar injuries.





A



B

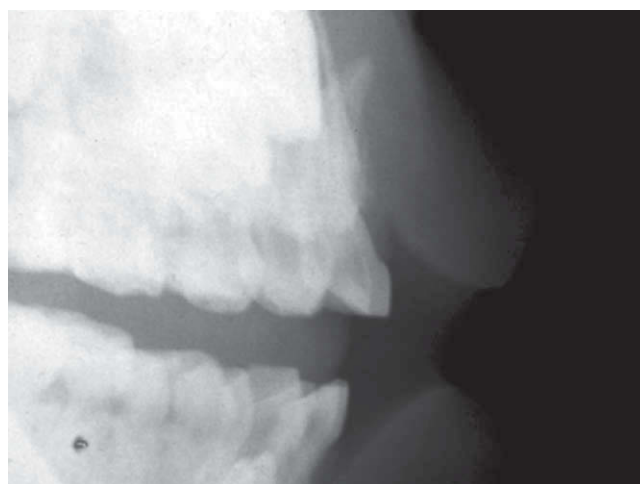


C

**Figure 7-3** **A**, Panoramic radiograph of a 12-year-old child after a bicycle accident with maxillary and mandibular anterior dento-alveolar fractures demonstrating displacement and loss of several permanent teeth. **B**, Left maxillary alveolar fracture; note the step in the occlusal plane. **C**, Maxillary alveolar fracture accompanied by luxation of both central incisors and left lateral incisor demonstrated by an increase in PDL space apically and laterally.



A



B

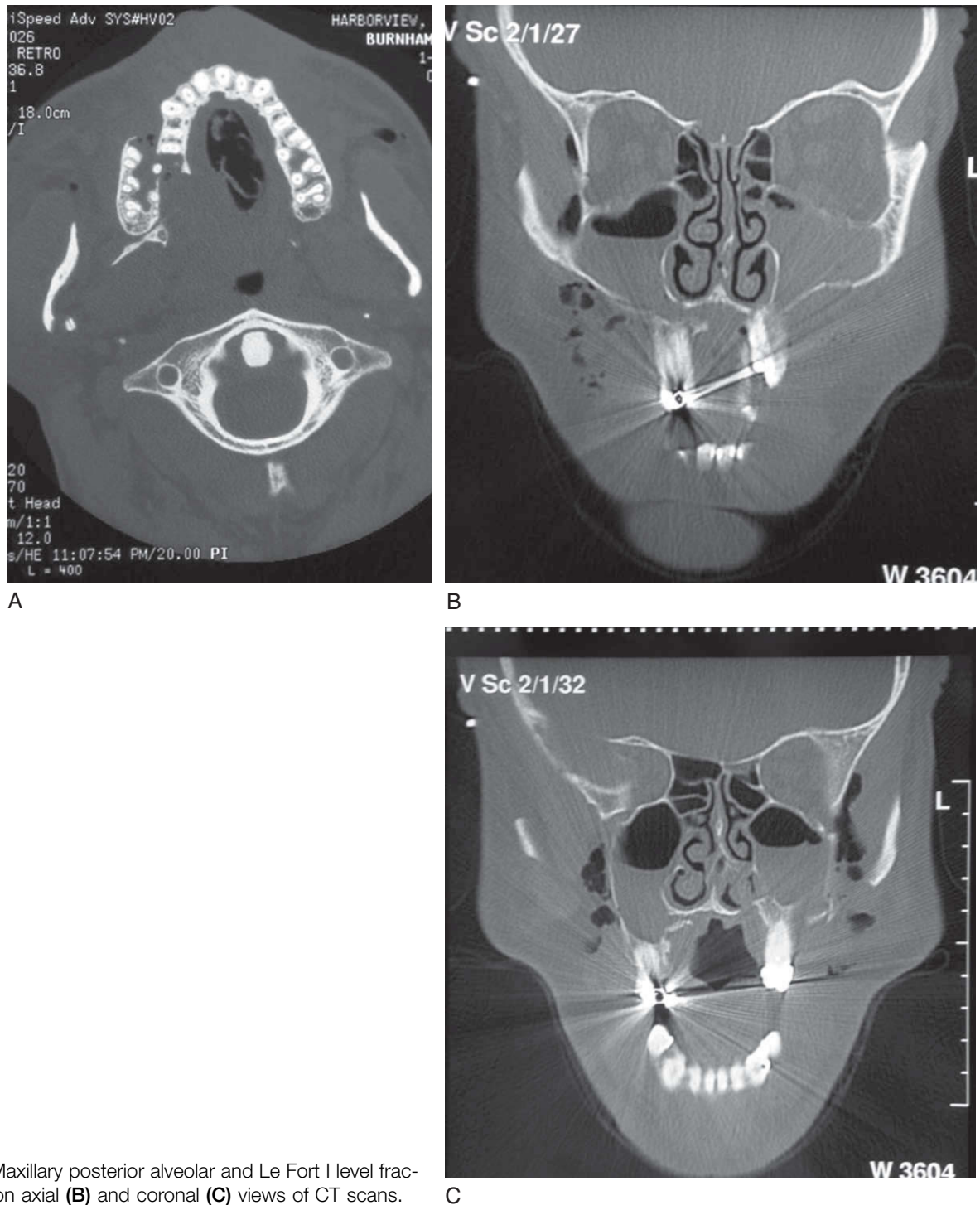
**Figure 7-4** **A**, Occlusal radiograph showing intrusion of primary tooth. **B**, Lateral radiograph in a child showing intrusion of primary tooth and perforation of labial cortical plate, which would be an indication for extraction.

## TREATMENT

At the time of the emergency visit, there should first be a thorough assessment of the patient as described in detail in Chapter 2. Additionally, in Box 7-1 at the end of this chapter, the reader will find protocols for the evaluation and treatment-planning assessment.

### ANTIBIOTIC TREATMENT AND TETANUS PROPHYLAXIS

Facial injuries and oral wounds are often contaminated with grass, gravel, glass, or soil. Because many of these fractures are open to these contaminants and microorganisms, there is an increased risk of infection. Antibiotics and tetanus prophylaxis should therefore be considered in the manage-



**Figure 7-5** A, Maxillary posterior alveolar and Le Fort I level fractures detected on axial (B) and coronal (C) views of CT scans.

ment of these injuries. Broad spectrum antibiotics, such as penicillin or clindamycin, also effective against gram-positive organisms, are recommended.<sup>39,48</sup>

If an individual has been previously immunized for tetanus within 10 years, then 0.5 ml of absorbed tetanus toxoid vaccine should be administered as a “booster” for any tetanus-prone or contaminated wounds. However, if more than 10 years have elapsed since the last tetanus inoculation,

then the patient should receive 0.5 ml of the tetanus toxoid for both tetanus-prone and for *all* puncture, open, clean, and minor wounds. There is no urgency for the administration of tetanus toxoid in the acute setting because it provides protection against the next injury and not the current injury. Tetanus immunoglobulin should be reserved for wounded patients who have never received a primary immunization against tetanus.<sup>42</sup>

The basic principles of the management of alveolar fractures are similar to the management of fractured bones elsewhere in the body:

- Reduction
- Fixation and immobilization
- Rehabilitation

The successful management of alveolar fractures involves immediate and accurate reduction and anatomical repositioning of the displaced alveolar fragment.<sup>8</sup> Reduction should be followed by fixation and immobilization of the reduced alveolar segment for a period of 4 to 6 weeks to achieve bony union.<sup>1</sup> The teeth in the fractured segment should be assessed appropriately during the immediate post-fixation period and then periodically for up to 5 years to determine the need for endodontic treatment.<sup>42</sup> Chapters 3 to 6 describe the clinical evaluation and management of the involved teeth.

## REDUCTION

The majority of the alveolar fractures can be reduced by simple manual repositioning under local anesthesia, either in a hospital emergency room or in a dental office. For the management of extensive intraoral and extraoral soft and hard tissue injuries, general anesthesia may be indicated, especially for patients with high anxiety and low compliance. These injuries can sometimes be complicated by the presence of luxated or extruded teeth, which may interfere with the proper reduction of the fractured segment. Therefore luxated teeth may need to be repositioned before reducing the alveolar fracture. Often to reposition the teeth, débridement of the wound or avulsion socket may first be necessary, removing any bone or tooth fragments, or coagulated blood in the socket (see Chapter 6). Every attempt should be made to preserve any portion of the alveolus and soft tissue that has a chance of survival. This will help to maintain the alveolar ridge height and width to help facilitate the future rehabilitation of the traumatized area (Fig. 7-6).

## FIXATION AND IMMOBILIZATION

Unlike fixation for avulsed teeth, fixation for alveolar fractures must be a rigid design to allow the bone to heal. If there are luxated teeth in the fractured alveolar segment, then the splint should allow physiological movement of these teeth while maintaining rigid fixation of the alveolar segment<sup>1</sup> (see Chapters 5 and 6). Stabilization and fixation of the reduced segment can be achieved by interdental wiring with arch bars, composite splints, acrylic splints, or occasionally plate-and-screw fixation. The choice of the type of splint depends on the type of alveolar fracture; the number of teeth present

in the arch; the presence of primary, permanent, or mixed dentition; and the location of the fracture.

Well-designed splints should:

- be easy to apply and remove
- provide adequate fixation for the entire period of immobilization
- allow easy endodontic access (if necessary)
- not interfere with occlusion
- not cause damage to the adjacent gingival tissue and teeth

Sometimes splints have to be modified in the mixed dentition phase or in partially edentulous patients, when there may not be enough teeth on each side of the fractured segment to adequately stabilize the fracture.

The period of immobilization can vary between 4 and 6 weeks. The key to adequate immobilization is to anchor the fracture segment rigidly to at least 3 to 4 stable teeth on both sides of the fractured alveolar segment. In some cases, maxillomandibular/intermaxillary fixation (MMF/IMF) may be necessary. MMF/IMF involves wiring the maxillary and mandibular arches together, after establishing proper occlusion, to avoid any movement of the segment during healing (Fig. 7-7)<sup>1</sup>.

## Composite Retained Wire Splint

A heavy stainless steel wire (19 or 22 gauge) can be adapted and rigidly secured with composite to at least three adjacent stable teeth on both sides of the fracture. This is a simple and effective method of stabilizing alveolar fractures and can be readily provided in a dental office (Fig. 7-8). Unlike splints for luxated or avulsed teeth that are passive (i.e., allowing slight movement), these splints have to be rigid during the period when the bone is healing. The advantage of the composite splint is that it helps to maintain oral hygiene and periodontal health during the period of fixation; it is easily applied and allows for endodontic access if necessary.

## Arch Bars

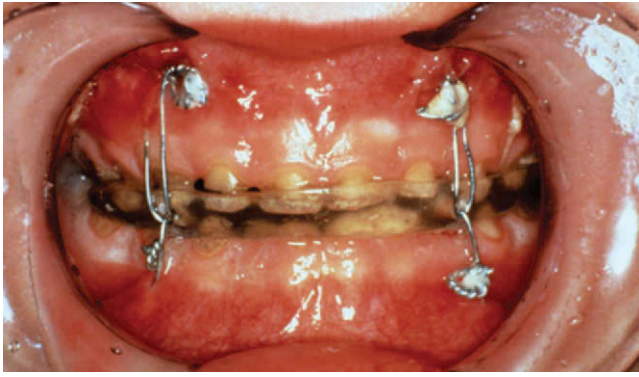
Several types of arch bars, including the recent light cure/bonded arch bars (Fig. 7-9), are available and typically used in hospital emergency rooms. Once the patient is adequately anesthetized and the fracture is reduced, the arch bar is applied to the facial surface of the teeth and secured with 25/24-gauge prestretched stainless steel wire threaded circumferentially through the interproximal spaces. By contrast, the *bonded* arch bars consist of hooks on a prefabricated malleable mesh backing that is attached to the teeth with light curing composite.<sup>11</sup> The clinician should be mindful to avoid inadvertent dislocation of a luxated tooth while securing the arch bar with wire. Avulsed incisors should be secured after replantation with the wire *coronal* to the cingulum to maintain the tooth in the socket rather than extruding it. The details of splinting luxated and avulsed teeth are described in Chapters 5 and 6, respectively.





**Figure 7-6** A to C, Posttraumatic deformity. Malocclusion after maxillary anterior dento-alveolar and mandibular fracture and loss of teeth. D, Loss of anterior maxillary teeth and alveolar bone. E and F, Malocclusion caused by intrusion of maxillary canines.





A



B

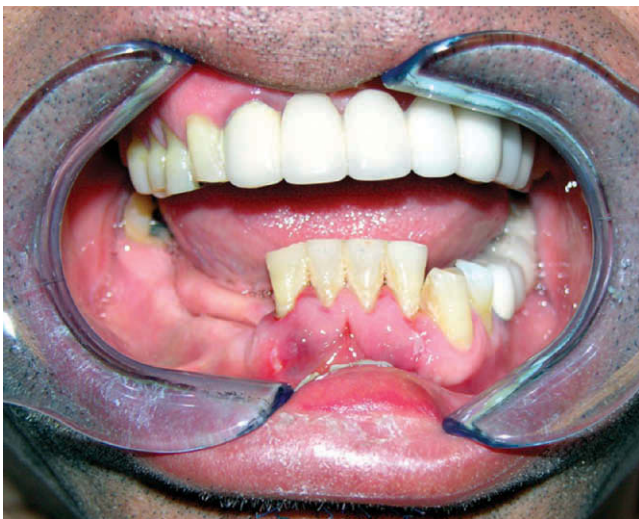
**Figure 7-7** A and B, Maxillo-mandibular fixation.



A



B



C



D

**Figure 7-8** A 45-year-old male was involved in an altercation and sustained a mandibular dento-alveolar fracture. **A**, He is unable to bring his teeth together due to the occlusal interference. **B**, Displacement of the fracture is evident on the panoramic radiograph. **C**, Mandibular dento-alveolar fracture was reduced under local anesthesia. **D**, Manual repositioning of the alveolar segment.





E



F

**Figure 7-8 cont'd, E,** After reduction the occlusion was checked. **F,** Fracture was fixed with a composite splint.



A



B



C

**Figure 7-9** Fixation with arch bars: **A** and **B,** Maxillary dento-alveolar fracture immobilized with arch bars and stainless steel wires. **C,** Use of bonded arch bars secured with light cured resin.

### Acrylic Splint

A simple acrylic splint can be fabricated and wired or cemented in place. First an impression is taken of both arches, and plaster models are fabricated. Next the fractured segment is cut out and repositioned *on the model*, verifying the proper occlusion with a model of the opposing arch. Then an acrylic splint is fabricated on the model with the

arch in this reduced position. The splint is now clinically used as a template for repositioning the fractured segment; it can be wired, cemented, or screwed in place (Fig. 7-10).

### Orthodontic Appliances

Orthodontic arch wires and edgewise brackets may be used for immobilizing dento-alveolar fractures. The arch wire





A



B

**Figure 7-10** A and B, Acrylic splints can be used for fixation during the mixed dentition.

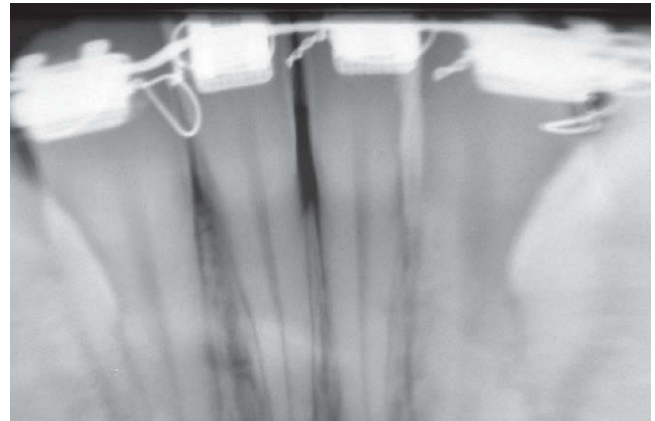
must be passive to prevent inadvertent orthodontic forces, which could create undesired tooth movement. These splints can be cleansed easily and can be easily applied in the outpatient dental setting (Fig. 7-11).

### Plate-and-Screw Fixation

Direct osteosynthesis with miniplates/microplates can be useful, especially in the management of palatal and anterior maxillary fractures.<sup>28</sup> The need for immobilization of the fracture by wiring the jaws together (maxillo-mandibular fixation) can be eliminated by using these direct fixation techniques. It also allows management of avulsed or luxated teeth independent of the alveolar fracture fixation. The surgical technique involves cleaning the wound, administration of local anesthesia, and exposing the fracture site via a mucosal incision or through a preexisting soft tissue laceration. The fracture is reduced manually with finger pressure, verifying proper arch alignment and occlusion. With large maxillary segment fractures, maxillo-mandibular fixation may be necessary intraoperatively to establish the arch alignment and occlusion, which can be removed after



A



B

**Figure 7-11** Splinting of dento-alveolar fracture using passive arch wire applied with orthodontic brackets and composite.

the fracture is reduced and fixed with titanium microplates and screws (Figs. 7-12 and 7-13).

Fractures of the alveolar bone that involve the floor of the maxillary sinus or tuberosity, with or without teeth, should be treated by repositioning the fragment and immobilizing it until it heals. If the alveolar segment is inadvertently removed, then the soft tissue repair of the defect must be carried out immediately to prevent an oral-antral communication.<sup>12,44</sup>

## REHABILITATION

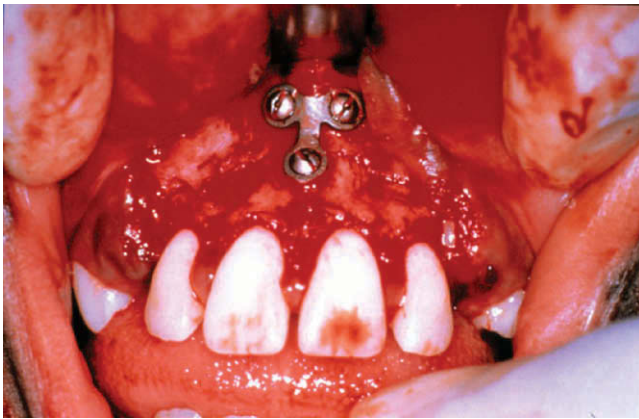
Alveolar fractures can result in the loss of teeth, bone, and adjacent soft tissue. Loss of anterior teeth can be devastating psychologically for adults and children because of the resulting esthetic and functional deficit.<sup>43,45</sup> The teeth lost as a result of injury can be replaced by a removable or fixed prosthesis, or by endosseous implants if there is adequate bone remaining. When tooth loss is accompanied with significant



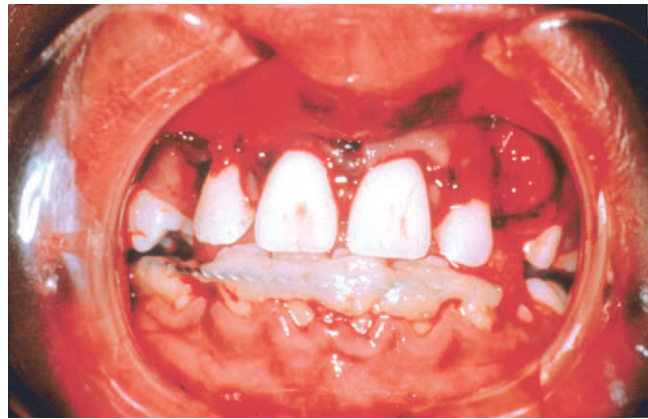
A



B



C



D

**Figure 7-12** A 12-year-old boy sustained maxillary and mandibular anterior alveolar fractures after a fall. **A**, Immediately after injury. **B**, Three months after fractures were treated. **C**, Maxillary anterior alveolar fracture-fixation with direct osteosynthesis. **D**, Mandibular anterior alveolar fracture was fixed and immobilized with composite wire splint.

alveolar bone loss, the residual defect may be difficult to restore. These defects often require alveolar ridge augmentation and soft tissue grafting before placement of endosseous implants; this will help assure implant success and achieve desirable esthetic and periodontal results. Alveolar ridge augmentation can be achieved by autogenous onlay or inlay grafting or by distraction of the residual bone (Fig. 7-14).<sup>38,43,45</sup>

## HEALING

When the force generated in a traumatic injury is greater than the inherent strength of bone, a fracture occurs. Unlike other tissues, which often respond to injury by the

formation of a scar, bone has the capacity to heal through actual bony regeneration.<sup>3,46</sup> This property allows the injured bone to regain its preinjury form, function, and strength.

## SEQUENCE OF FRACTURE HEALING

When bone is fractured, a progression of tissue responses occurs that is designed to:

- Remove necrotic and damaged tissue components (the *inflammatory* phase)
- Reestablish the vascular supply (the *reparative* phase)
- Produce a new skeletal matrix (the *remodeling* phase)<sup>29</sup>

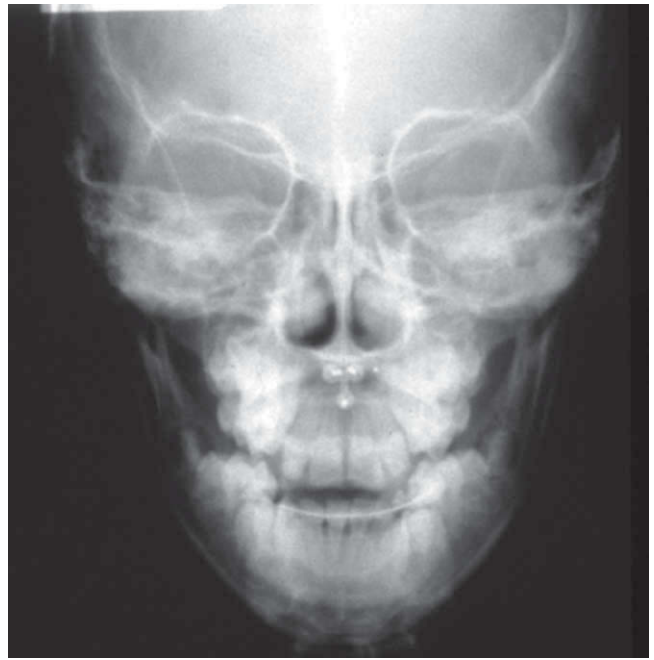
The *inflammatory* phase occurs over the first 24 to 48 hours, beginning with bleeding from the adjacent injured

*Text continued on p. 142*





A



B

**Figure 7-13** A and B, Radiographs of case from Figure 7-12. Posteroanterior and lateral radiographs after fixation with direct osteosynthesis and composite splint.



A

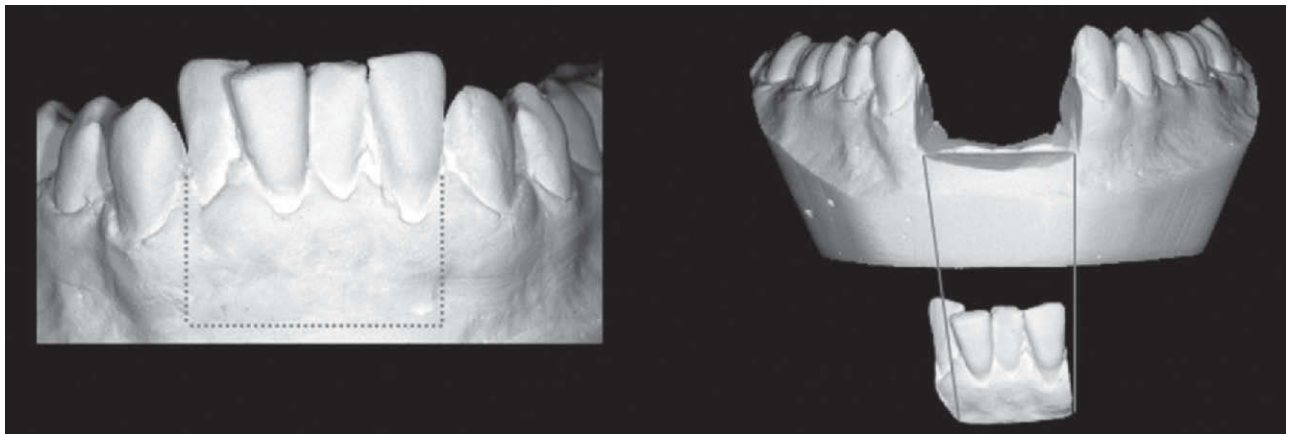


B

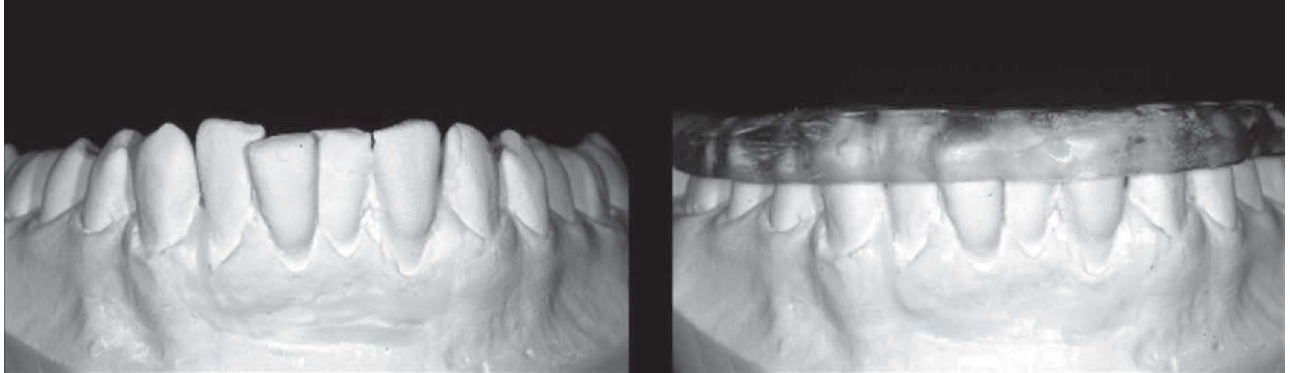


**Figure 7-14** A 38-year-old female who sustained maxillary and mandibular alveolar fractures after a fall from a cliff. Treatment was delayed, resulting in a posttraumatic deformity. **A**, Lack of the upper lip support caused by loss of anterior maxillary teeth. **B**, Malocclusion due to nonreduced mandibular alveolar fracture.

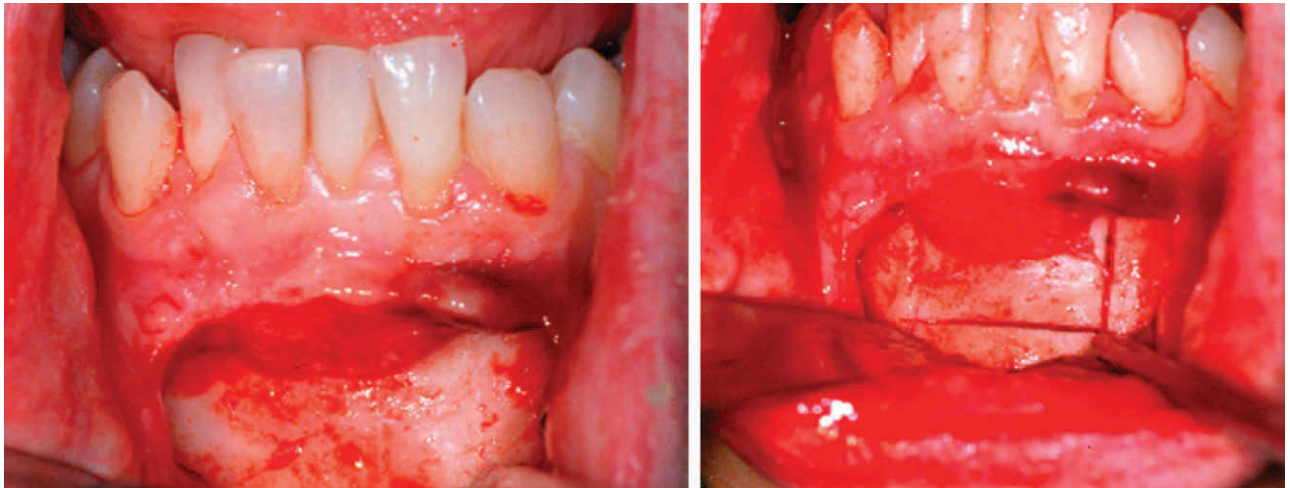




C



D



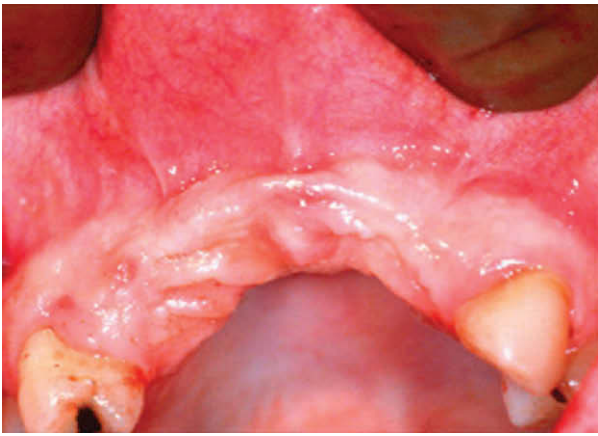
E



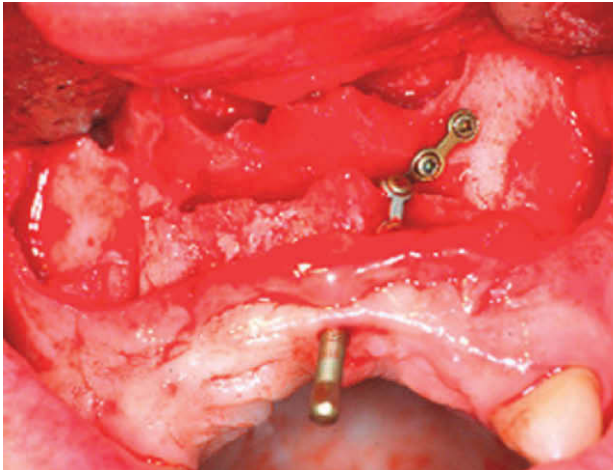
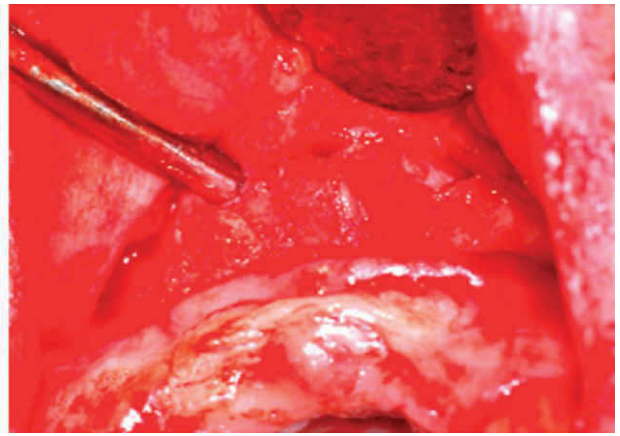
F

**Figure 7-14, cont'd** C, Model surgery of segmental mandibular osteotomy to reposition alveolar segment. D, Model surgery completed and acrylic splint fabricated. E, Segmental mandibular alveolar osteotomy, demonstrating the cuts in between the roots of the teeth. F, The osteotomized alveolar segment was repositioned and secured by rigid fixation (bone screw).

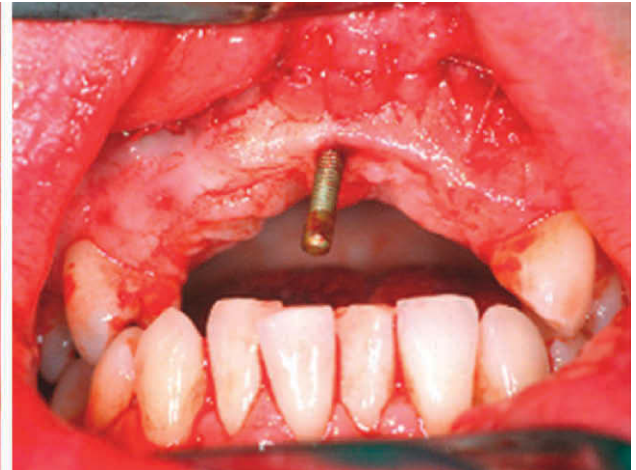
*Continued*



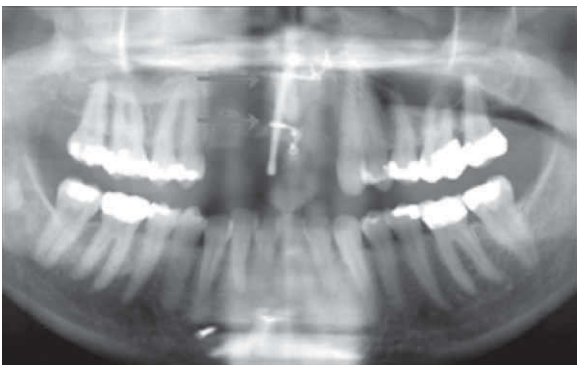
G



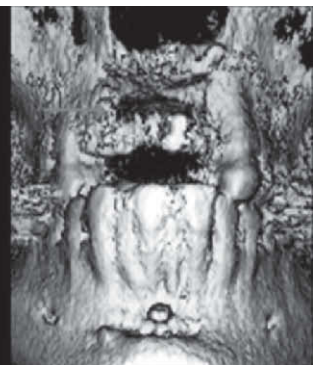
H



I

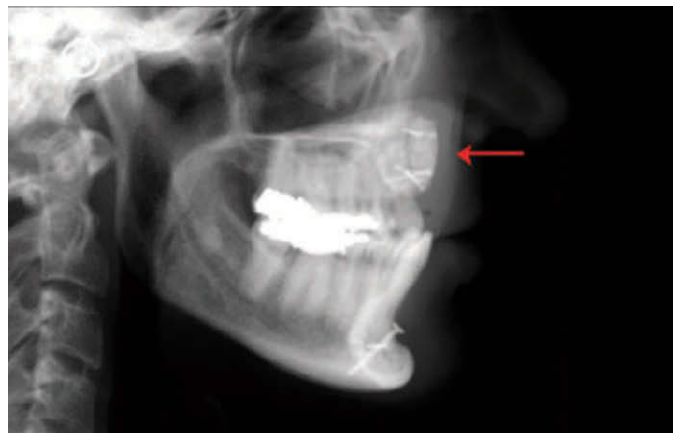


J



**Figure 7-14, cont'd** G, Segmental maxillary osteotomy. H, Placement of alveolar distractor. I, Completion of alveolar distraction. J, Maxillary alveolar ridge augmentation included alveolar distraction.

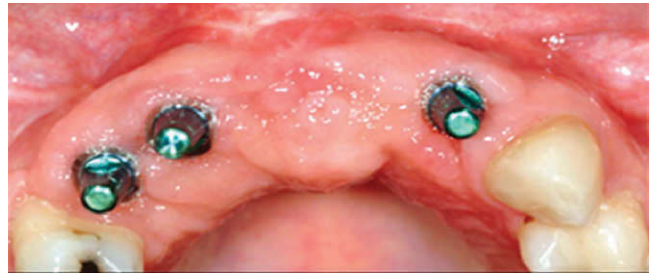




K



L



M



N



O



P



**Figure 7-14, cont'd K**, Additional augmentation with only grafting was necessary even after alveolar distraction. **L and M**, Three maxillary implants placed to support an implant-retained bridge. **N, O, and P**, Esthetics and function restored by complete prosthetic rehabilitation.



bone and soft tissue, and clot formation at the fracture site. This compromises the blood supply to the bone and results in the death of osteocytes and the ingress of osteoclasts. There is active phagocytosis and lysis by monocytes and osteoclasts, and the subsequent formation of granulation tissue.

In the *reparative* phase, occurring 4 to 40 days after the injury, increased cellular proliferation produces osteoid and cartilage, or *callus*, along the inner and outer aspect of the fractured bone. The callus becomes hard by endochondral ossification and mineralization of the immature bone.<sup>29,32</sup>

Finally, during the remodeling phase, occurring 40 to 140 days after the injury, immature woven bone is replaced by lamellar bone. By the end of this phase, bone regains its preinjury strength and shape. Primary bone healing occurs when there is a good reduction with direct bone segment contact and approximation, with minimal or no mobility at the fracture site. This can be achieved by open reduction and rigid internal fixation. In secondary bone healing, there is an intermediate fibro-cartilaginous callus formed within the fracture gap, which is subsequently converted to bone.

Healing of alveolar fractures may be adversely influenced by:

- Poor nutrition
- Compromised immune status
- Endocrine disorders (such as diabetes)
- Traumatic occlusion
- Inadequate fixation of the fractured segment

Additionally, poor reduction may result in the entrapment of soft tissue between the bone margins, enabling infection to develop at the fracture site.

## COMPLICATIONS

Alveolar fractures cause injury not only to the supporting bone, but also potentially to the pulp and PDL of the involved teeth, and the adjacent mucogingival soft tissue. The complications that arise from alveolar fractures are a direct consequence of damage to any of these tissues and inadequate treatment.

### MALOCCLUSION

Inaccurate reduction of the alveolar fracture can result in malocclusion. If this problem is recognized early, then it can be repositioned accurately and immobilized; however, repositioning the fragment after it is healed is difficult without surgical or orthodontic intervention. An osteotomy of the alveolar segment may be necessary to properly reposition the segments (see Fig. 7-6). In some cases, orthodontic tooth movement alone may correct the malocclusion problem.



**Figure 7-15** Poor reduction and fixation of maxillary alveolar fracture with arch bars can result in loss of teeth and bone.

### LOSS OF ALVEOLAR BONE

Insecure fixation can cause mobility of the segment, and eventually small alveolar segments can sequester and result in the loss of bone and teeth. Therefore proper fixation and an adequate period of immobilization is necessary to allow the bone to heal (Fig. 7-15).

### LOSS OF TEETH

Loss of teeth in alveolar fractures can occur as a result of severe comminution of the bony socket, fractured and non-repairable roots, or subsequent root resorption. Loss of teeth can have devastating psychological implications, especially if these are anterior teeth.<sup>15,17</sup> Early recognition of clinical and radiographic signs of adverse pulpal and periodontal changes is essential;<sup>16</sup> proper management is imperative.

## ENDODONTIC IMPLICATIONS

Teeth within the fractured alveolar segment show varied responses to injury, including reversible pulpitis, irreversible pulpitis, canal calcification, pulp necrosis, and root resorption. The most detrimental sequelae to teeth are pulp necrosis and resorption.<sup>37</sup> Investigators have evaluated the prognosis of permanent teeth in the line of mandibular fractures.<sup>36</sup> They found that pulp necrosis was more frequent in cases in which the fracture line ran through the apex or when there is a dislocation of the fracture segment. When the alveolar fracture is apical to the root tips or when the apices of teeth are open, the vascular supply to the teeth is less at risk. Another study has also shown that a minor luxation, an intact crown, and immaturity of the root positively influence pulpal and periodontal healing.<sup>20</sup>

## PROGNOSIS AND OUTCOME ASSESSMENT

Alveolar fractures can generally be attended to quickly in the emergency room and reduction can be easily accomplished under local anesthesia. However, when they occur as part of more extensive injuries, the importance of early intervention of these alveolar fractures is often ignored because of the urgency of other critical injuries.<sup>13</sup>

The prognosis of alveolar fractures depends on the following factors:

- Time interval between injury and fixation of alveolar fracture
- Type of alveolar fracture
- Associated dental injury (luxation or avulsion) to the teeth in the fractured segment
- Stage of root development of the teeth involved
- The health of the periodontal tissues
- The presence of significant comminution of bone and teeth

Delay of treatment directly affects the prognosis of the teeth involved in the fracture. Pulp necrosis and root resorption are consequences of dento-alveolar injuries often caused by delay in treatment or inadequate treatment.<sup>8</sup> Andreasen<sup>7</sup> summarized the occurrence of tooth pathosis associated with permanent teeth involved in fractures of the alveolar process as:

- Pulp necrosis: 75%
- Pulp canal obliteration: 15%
- Progressive root resorption: 11%
- Loss of marginal bone support: 13%

## PEDIATRIC CONSIDERATIONS

Children are anatomically, physiologically, and psychologically different from adults. An understanding of the anatomy and growth of the facial skeleton, development of teeth and alveolus, eruption and occlusion, and psychological development is essential in the management of facial and dento-alveolar injuries. In children, nasal bone and dento-alveolar fractures are the most common injuries of the facial skeleton, but they are often treated in the outpatient setting and not recorded in the hospital database of maxillofacial injuries (Fig. 7-16).<sup>14,27,50</sup> Unlike these injuries, mandibular fractures are more commonly seen in hospital settings and account for 56% of facial fractures (Fig. 7-17).<sup>18,25</sup> In children, falls on stairs, bicycles, sports-related accidents (e.g., blunt trauma from baseball, cricket bats, or hockey sticks), and vehicular accidents account for the majority of facial trauma; less common causes include child abuse and neglect.<sup>25,35</sup> These traumatic injuries commonly occur between the ages of 2 and 4 and 8 and 10 years.<sup>19</sup>

In young children with primary dentition, luxation injuries of the teeth, particularly intrusions and avulsions, are more common than alveolar fractures.<sup>21</sup> If alveolar fractures occur before the eruption of permanent teeth, they are minimally displaced and rarely require stabilization or fixation after reduction because the bone heals so rapidly (Fig. 7-18).<sup>4</sup> This is because the tooth-to-bone ratio is low and teeth are developing. The bone is more elastic with a higher ratio of cancellous to cortical bone. In addition, the jaws are relatively small and not yet developed, compared with the cranium.<sup>14,27</sup> Unlike the primary dentition phase, the mixed dentition has a higher tooth-to-bone ratio, which makes the alveolus more prone to fractures through the tooth crypts.<sup>27</sup>

Dento-alveolar injuries in children can often cause the parents to panic at the sight of blood, loose teeth, and debris in the mouth, causing more emotional trauma to the child, and making management of the injury more difficult. These injuries may often require evaluation and treatment under general anesthetic. The presence of loose primary teeth and a mixed dentition makes the fixation and stabilization of these injuries difficult. It may be necessary to fabricate an acrylic splint in these cases or apply a modified wire splint (Fig. 7-19).

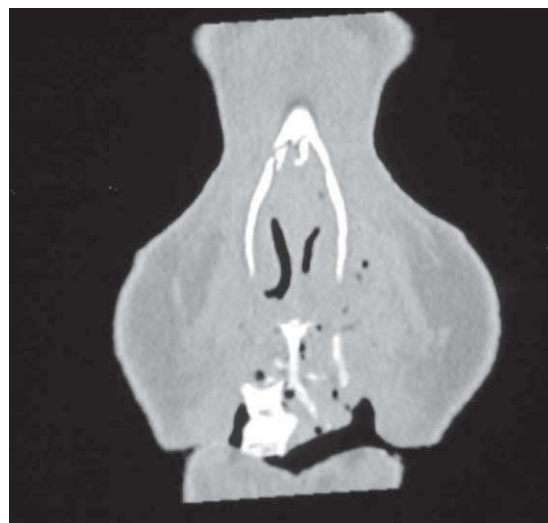
Alveolar fractures or luxation injuries of the primary dentition can affect the developing permanent tooth buds.<sup>47</sup> The effect on the underlying permanent tooth is dependent upon its stage of development and the extent of the injury to the primary tooth. This is especially true with intrusion and avulsion injuries of primary teeth in children between 2 and 3 years of age; these injuries have the most frequent and deleterious effects on the developing permanent dentition.<sup>10</sup> Dento-alveolar injuries may cause subsequent trauma to developing permanent teeth or tooth buds,<sup>9,30</sup> and may include:

- Discoloration of the crown, with or without enamel hypoplasia
- Cessation of root development
- Dilaceration of the root
- Malformation of the crown
- Odontoma-like formation
- Disturbance in eruption

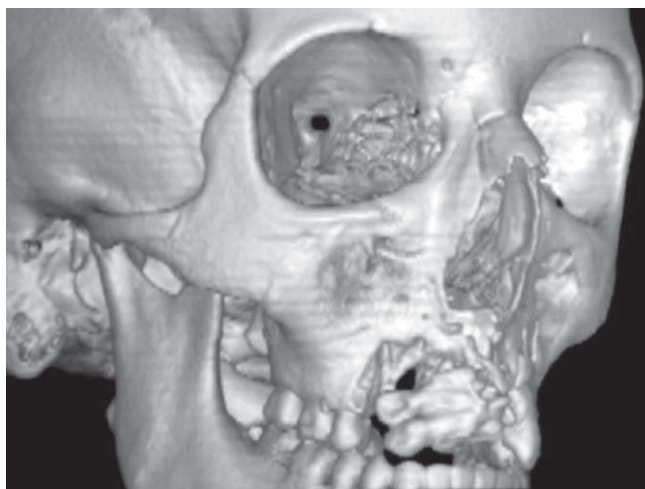
It is important to recognize when to remove a primary tooth to prevent deleterious effects on the developing permanent tooth. In case of intrusion and luxation injuries, if there is perforation of the labial cortical plate on the lateral film, then the primary tooth should be removed. If there is any question of infection of the primary tooth during the early formative years of the permanent dentition, then it is prudent to initiate endodontic treatment or remove the primary tooth to prevent deleterious effects on the developing permanent tooth bud.<sup>49</sup> Injured primary teeth must be reevaluated periodically; the permanent teeth in these young patients must be monitored with respect to their development and eruption.



A



B



C



D



E



F

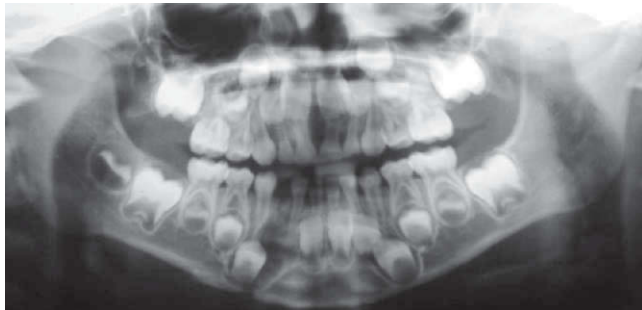




A



B



C

**Figure 7-17** **A**, Five-year old boy kicked by a horse sustained a mandibular fracture. **B**, Sequestration of the permanent lateral mandibular incisor from the line of fracture. **C**, Right parasymphysis mandibular fracture observed on panoramic radiograph.



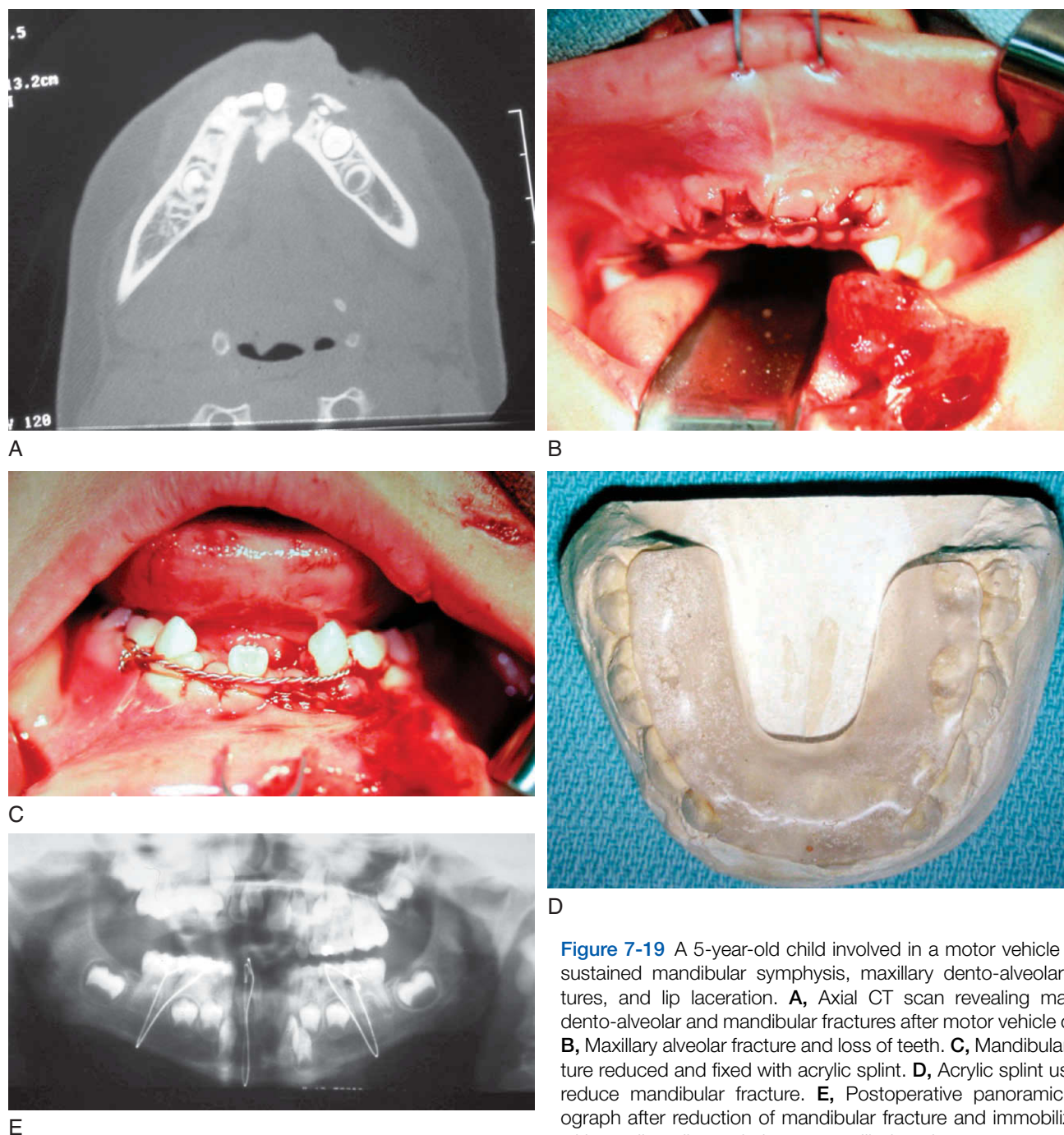
A



B

**Figure 7-18** **A**, A 5-year-old child after a fall; maxillary anterior alveolar fracture caused by entrapment in a rope fence. **B**, Anterior maxillary alveolar fracture was reduced by manual repositioning under general anesthesia.

**Figure 7-16** A 14-year-old female sustained severe maxillary alveolar and nasal bone fractures after a fall. **A** and **B**, Coronal views of the maxillofacial CT scan reveal alveolar and nasal bone fractures. **C** and **D**, These three dimensional views of the CT scan give an idea of the comminution and the displacement of the alveolar fracture and loss of anterior maxillary teeth. **E**, Six months after injury without temporary prosthesis. **F**, Six months after injury with temporary prosthesis. She will have further alveolar ridge augmentation and fixed implant-retained prosthesis after growth is completed.



**Figure 7-19** A 5-year-old child involved in a motor vehicle crash sustained mandibular symphysis, maxillary dento-alveolar fractures, and lip laceration. **A**, Axial CT scan revealing maxillary dento-alveolar and mandibular fractures after motor vehicle crash. **B**, Maxillary alveolar fracture and loss of teeth. **C**, Mandibular fracture reduced and fixed with acrylic splint. **D**, Acrylic splint used to reduce mandibular fracture. **E**, Postoperative panoramic radiograph after reduction of mandibular fracture and immobilization with acrylic splint and circum-mandibular wires.

## CONCLUSION

Alveolar fractures can be variable in their presentation with regard to location and severity of occurrence. Because they can happen secondary to any facial trauma, their detection and management are imperative. Associated teeth must be

properly evaluated and treated as necessary to assure the successful healing of the fracture site and to assure the viability of the involved teeth. The clinician should be aware that other injuries may have also been incurred secondary to the alveolar fracture and that a proper assessment of the trauma is essential.



**BOX 7-1****Summary: Evaluation and Management of Alveolar Fractures****Diagnosis**

- Inspect the injured dento-alveolar segment for arch discontinuity.
- Check the soft tissue wounds for debris, foreign bodies, hematoma, and loss of tissue.
- Check for any mobility of alveolar segment and/or occlusal interferences.
- Assess any injury to the teeth with regard to fractures, luxations, or avulsions.

**Radiographic Examination**

- Expose, as necessary, occlusal, periapical, and panoramic views.
- Observe for thickening of the PDL space, with an increased space laterally or apically indicating luxation or displacement of teeth.
- Evaluate for fractures with or without significant separation of the fragment; horizontal fracture lines are usually above or at the same level of the apices of the teeth; vertical fracture lines usually run parallel to the PDLs of the teeth.

**Management**

- Reassure and sedate the patient as necessary.
- Adequate local anesthesia with block or local infiltration; occasionally, intravenous sedation or general anesthesia may be necessary in young children and extremely anxious patients.
- Mobilize the segment digitally and verify occlusal relationship, reduce the fragment by repositioning it.
- Immobilize the segment in the reduced position by application of a bonded splint or an arch bar; consider maxillo-mandibular fixation for large segments of alveolar fracture.
- Relieve occlusal interferences when maxillo-mandibular fixation is not used; maintain splint for 4 to 6 weeks.
- Do not extract teeth from the segment unless danger of aspiration exists.

**Supportive Treatment**

- Prescriptions for adequate analgesia
- Antibiotic and tetanus-prevention therapy
- Instructions to apply ice and diet restrictions, soft and liquid diet for 4 weeks
- Oral hygiene instructions

**Immediate Follow-Up**

- An immediate radiograph should be obtained to verify the position of the teeth and the alveolar segment.
- Have the patient return for follow-up in 1 week.
- Observe the condition of the healing soft tissue.
- Ensure that pain control is adequate.
- Involved teeth require initial endodontic evaluation.

**Long-Term Follow-Up**

- Remove the splint in 4 to 6 weeks.
- Evaluate stability of the alveolar segment and teeth.
- Obtain radiographs and complete an endodontic and periodontal assessment.
- Augmentation of the alveolar ridge.
- Extraction and replacement of nonrestorable teeth or teeth with a poor prognosis.

**REFERENCES**

1. AAE: Recommended guidelines by American Association of Endodontists for treatment of traumatic dental injuries, 2003.
2. Abubaker AO, Giglio JA, Mourino AP: Diagnosis and management of dentoalveolar injuries. In Fonseca RJ, editor: *Textbook of oral and maxillofacial surgery*, ed 1, Philadelphia, 2000, WB Saunders.
3. Adzick NS: Wound healing biologic and clinical features. In Sabiston DC, editor: *Textbook of surgery: the biological basis of modern surgical practice*, ed 15, Philadelphia, 1997, WB Saunders.
4. Amaratunga NA: The relation of age to the immobilization period required for healing of mandibular fractures, *J Oral Maxillofac Surg* 45(2):111-113, 1987.
5. Andreasen JO: Fractures of the alveolar process of the jaw. A clinical and radiographic follow-up study, *Scand J Dent Res* 78(3):263-272, 1970.
6. Andreasen JO: Classification, etiology and epidemiology. In Andreasen JO, editor: *Traumatic injuries of the teeth*, ed 3, Copenhagen, 1994, Munksgaard.
7. Andreasen JO: Injuries to the supporting bone. *Textbook and color atlas of traumatic injuries to the teeth*, ed 3, Copenhagen, 1994, Munksgaard.
8. Andreasen JO, Andreasen FM, Skeie A et al: Effect of treatment delay upon pulp and periodontal healing of traumatic dental injuries—a review article, *Dent Traumatol* 18(3):116-128, 2002.
9. Andreasen JO, Ravin JJ: Enamel changes in permanent teeth after trauma to their primary predecessors, *Scand J Dent Res* 81(3):203-209, 1973.
10. Andreasen JO, Sundstrom B, Ravn JJ: The effect of traumatic injuries to primary teeth on their permanent successors. I.



- A clinical and histologic study of 117 injured permanent teeth, *Scand J Dent Res* 79(4):219-283, 1971.
11. Baliga M, Shenoy V, Krishna L: Quick technique for immobilisation of dentoalveolar fracture, *Br J Oral Maxillofac Surg* 41(6):417, 2003.
  12. Banks P: *Killey's fractures of the middle third of the facial skeleton: dental practitioner handbook no. 3*, ed 4, Bristol, 1981, Wright PSG.
  13. Batstone MD, Waters C, Porter SA et al: Treatment delays in paediatric dento-alveolar trauma at a tertiary referral hospital, *Aust Dent J* 49(1):28-32, 2004.
  14. Baumann A, Troulis MJ, Kaban LB: Facial trauma II: Dentoalveolar injuries and mandibular fractures. In Kaban LB, Troulis MJ, editors: *Pediatric oral and maxillofacial surgery*, Philadelphia, 2004, Saunders.
  15. Berman CL, Berman GL: The psychological implications of tooth loss, *Dent Clin North Am* 13(4):961-966, 1969.
  16. Cohen S, Blanco L, Berman LH: Early radiographic diagnosis of inflammatory root resorption, *Gen Dent* 51(3):235-240, 2003.
  17. Davis DM, Fiske J, Scott B et al: The emotional effects of tooth loss: a preliminary quantitative study, *Br Dent J* 188(9):503-506, 2000.
  18. Dodson TB: Mandibular fractures in children, OMS knowledge update 1 (Part II), pp. 95-107, 1995.
  19. Dorney B: Inappropriate treatment of traumatic dental injuries, *Aust Endod J* 25(2):76-78, 1999.
  20. Ebeleseder KA, Santler G, Glockner K et al: An analysis of 58 traumatically intruded and surgically extruded permanent teeth, *Endod Dent Traumatol* 16(1):34-39, 2000.
  21. Flores MT: Traumatic injuries in the primary dentition, *Dent Traumatol* 18(6):287-298, 2002.
  22. Gassner R, Tuli T, Hachl O et al: Cranio-maxillofacial trauma: a 10 year review of 9,543 cases with 21,067 injuries, *J Craniomaxillofac Surg* 31(1):51-61, 2003.
  23. Hill CM, Crosher RF, Mason DA: Dental and facial injuries following sports accidents: a study of 130 patients, *Br J Oral Maxillofac Surg* 23(4):268-274, 1985.
  24. Hopkins R: Mandibular fractures: Treatment by closed reduction and indirect skeletal fixation. In Rowe NL, Williams JLL, editors: *Maxillofacial injuries*, ed 2, Edinburgh, 1994, Churchill Livingstone.
  25. Iida S, Matsuya T: Paediatric maxillofacial fractures: their aetiological characters and fracture patterns, *J Craniomaxillofac Surg* 30(4):237-241, 2002.
  26. Jarvinen S: Incisal overjet and traumatic injuries to upper permanent incisors. A retrospective study, *Acta Odontol Scand* 36(6):359-362, 1978.
  27. James D: Maxillofacial injuries in children. In Rowe NL, Williams JLL, editors: *Maxillofacial injuries*, ed 2, Edinburgh, 1994, Churchill Livingstone.
  28. Kanagaraja S, Kashani H, Zellin G: Fixation of comminuted alveolar ridge fracture after severe trauma by application of ti-osteosynthesis plates on soft tissue: a technical note, *J Oral Maxillofac Surg* 63(5):720-722, 2005.
  29. Kempers KG, Hender BH: Basic principles of treatment of hard and soft tissue injuries, In Fonseca RJ, editor: *Oral and maxillofacial surgery*, ed 1, Philadelphia, 2000, Saunders.
  30. Kenwood M, Seow WK: Sequelae of trauma to the primary dentition, *J Pedod* 13(3):230-238, 1989.
  31. Leathers RD, Gowans RE: *Management of alveolar and dental fractures. Principles of oral and maxillofacial surgery, part 4: maxillofacial trauma*, Hamilton, Ontario, 2004.
  32. Luyk NH: Principles of management of fractures of the mandible. In Peterson LJ, Indersano AT, Marciani RD et al: *Principles of oral and maxillofacial surgery*, Philadelphia, 1992, JB Lippincott.
  33. Margarone JE, Hall R: *Management of alveolar and dental fractures. Principles of oral and maxillofacial surgery*, ed 2, Philadelphia, 1997, Lippincott-Raven Publishers.
  34. Motamedi MH: An assessment of maxillofacial fractures: a 5-year study of 237 patients, *J Oral Maxillofac Surg* 61(1):61-64, 2003.
  35. Needleman HL: Orofacial trauma in child abuse: types, prevalence, management, and the dental profession's involvement, *Pediatr Dent* 8(1 Spec No):71-80, 1986.
  36. Oikarinen K, Lahti J, Raustia AM: Prognosis of permanent teeth in the line of mandibular fractures, *Endod Dent Traumatol* 6(4):177-182, 1990.
  37. Oulis C, Vadiakas G, Siskos G: Management of intrusive luxation injuries, *Endod Dent Traumatol* 12(3):113-119, 1996.
  38. Penchas J, Guerra LR, Block MS: Implant reconstruction of a facial trauma patient: clinical report, *Implant Dent* 3(3):176-178, 1994.
  39. Pogrel MA: Infection management in dento-alveolar surgery, *Oral and Maxillofac Surg Clin North Am* 5:127-135, 1993.
  40. Powers MP, Quershy FA: Diagnosis and management of dentoalveolar injuries. In Fonseca JR, Walker RV, Bets NJ et al, editors: *Oral and maxillofacial trauma*, ed 2, Philadelphia, 1997, WB Saunders.
  41. Ravn JJ: Dental injuries in Copenhagen schoolchildren, school years 1967-1972, *Community Dent Oral Epidemiol* 2(5):231-245, 1974.
  42. Rhee P, Nunley MK, Demetriades D et al: Tetanus and trauma: a review and recommendations, *J Trauma* 58(5):1082-1088, 2005.
  43. Sclaroff A, Elluru RG, Gay WD: Dental implantation for restoration of posttraumatic deformities: avulsion injuries, *Facial Plast Surg* 16(2):153-167, 2000.
  44. Sowray JH: Localised injuries to the teeth and the alveolar process. In Rowe NL, Williams JLL, editors: *Maxillofacial injuries*, ed 2, Edinburgh, 1994, Churchill Livingstone.
  45. Stevens MR, Heit JM, Kline SN et al: The use of osseointegrated implants in craniofacial trauma, *J Craniomaxillofac Trauma* 4(1):27-34, 1998.
  46. Szachowicz EH: Facial bone wound healing: an overview, *Otolaryngol Clin North Am* 28(5):865-880, 1995.
  47. von Gool AV: Injury to the permanent tooth germ after trauma to the deciduous predecessor, *Oral Surg Oral Med Oral Pathol* 35(1):2-12, 1973.
  48. Weitekamp MR, Caputo GM: Antibiotic prophylaxis: update on common clinical uses, *Am Fam Physician* 48(4):597-604, 1993.
  49. Wilson CF: Management of trauma to primary and developing teeth, *Dent Clin North Am* 39(1):133-167, 1995.
  50. Zachariades N, Papavassiliou D, Koumoura F: Fractures of the facial skeleton in children, *J Craniomaxillofac Surg* 18(4):151-153, 1990.

# SOFT TISSUE INJURIES AND MANAGEMENT



LUCIA BLANCO AND STEPHEN COHEN

## CHAPTER OUTLINE

EPIDEMIOLOGY OF SOFT TISSUE INJURIES  
 ETIOLOGY OF SOFT TISSUE INJURIES  
 MANAGEMENT OF SOFT TISSUE INJURIES  
 THE FACE  
     Immediate Treatment of Facial Trauma  
     Complementary Treatment for Facial Trauma  
 THE LIPS  
 CYANOACRYLATES AND THEIR DENTAL APPLICATIONS  
     Clinical Examination of the Lips  
     Radiographic Examination of the Lips  
 TECHNIQUE FOR CLOSING LIP WOUNDS WITH  
     CYANOACRYLATES  
     Advantages of Cyanoacrylates  
     Disadvantages of Cyanoacrylates  
 THE MUCOSA  
 THE LABIAL FRENUM  
 THE TONGUE  
 HEALING MECHANISM OF ORAL SOFT TISSUE WOUNDS  
 PREVENTIVE MEASURES TO AVOID SOFT TISSUE  
 INJURIES  
 CONCLUSION

Oral trauma may involve soft tissue, teeth, and bone. Soft tissue injuries present different and complex traumatic lesions.<sup>8,56,66,101,176</sup> The most common injuries to soft tissue are penetrating wounds, lacerations, contusions, abrasions, bruises, ecchymosis, traumatic ulcers, and hematomas. Traumatized patients can sustain one or more of these injuries simultaneously when severe trauma occurs<sup>38,176</sup> (Fig. 8-1).

The protocol for thorough and delicate management of soft tissue injuries is to provide appropriate emergency and follow-up treatment to minimize posttrauma sequelae, especially when the injury involves the face.<sup>38,86,101,139</sup>

For purposes of this discussion, paraoral and oral soft tissues include the following structures:

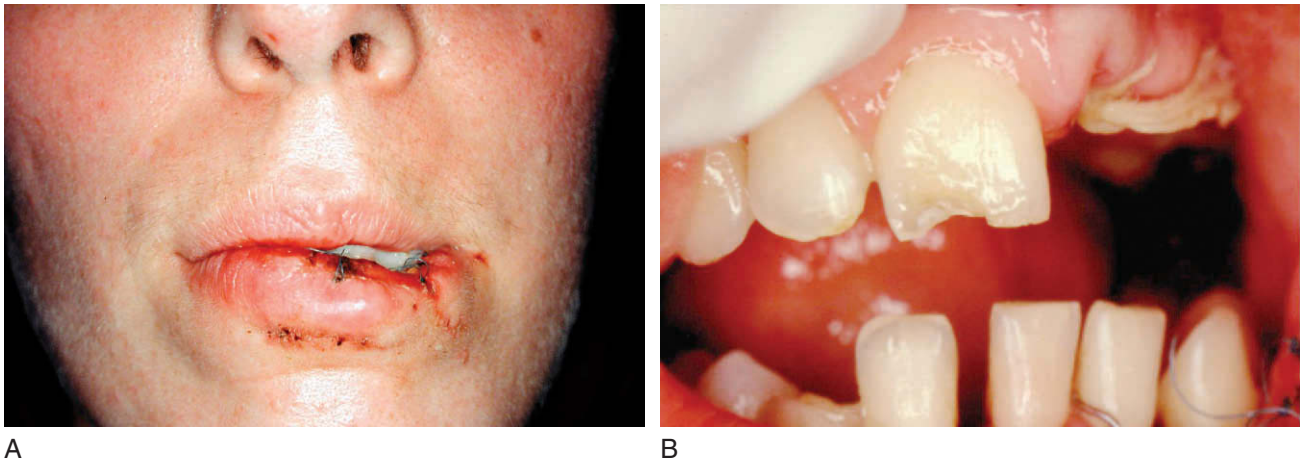
- Face
- Lips
- Mucosa (cheeks and periodontium)
- Labial frenum
- Tongue

## EPIDEMIOLOGY OF SOFT TISSUE INJURIES

According to Morad “epidemiology is an important and powerful tool in public health used to monitor health, observe trends, and identify risk factors for use in strategy, policy and planning of services and interventions.”<sup>114</sup>

Several epidemiological studies<sup>3,63</sup> have shown that the injuries in soft tissues are frequent and depend on the patient’s age. Statistics from childhood injuries are helpful in guiding pediatric prevention so that further possible traumas can be prevented.<sup>3</sup>

Males predominate through all age groups for all types of oral injuries.<sup>12,27,149</sup> Most soft tissue injuries are located between the nose and the chin area.<sup>180</sup>



**Figure 8-1** A 26-year-old woman who suffered a traffic injury. **A**, Ten days after trauma. The patient sustained several injuries simultaneously. **B**, She presented with avulsion of the maxillary left incisors, canine, premolars, and first molar. She also had an uncomplicated fracture of the maxillary right central incisor and canine (arrow) and avulsion of the mandibular right lateral incisor. Note the suture in the maxillary left posterior area.

School injuries, among others, make up the bulk of unintentional traumatic lesions.<sup>61,83</sup> Facial assault traumas have reached epidemic proportions. Although the biomechanics of injury, methods of repair, and general demography of blunt facial trauma are well known, statistical associations between epidemiological factors, incidence, and outcome are poorly understood.<sup>38</sup>

As researchers and clinicians are constantly working on new preventive and therapeutic measures to treat traumatic injuries, it is necessary to continue expanding our knowledge of the epidemiology of soft tissue trauma and provide adequate epidemiological surveillance.<sup>83,101,136,146</sup>

## ETIOLOGY OF SOFT TISSUE INJURIES

The nature of traumatic injuries to oral soft tissue varies according to the patient's age group.<sup>23,83,89,130,156</sup> Injuries are frequently triggered by a child's curiosity and most are preventable; they are most likely to occur when parents do not anticipate how risk behavior changes as their children grow older.<sup>3,164</sup> When a toddler begins to walk, falls are the most frequent causes of dental and soft tissue injuries because toddlers have immature equilibrium. In these early stages of life, most falls occur in and around home (Figs. 8-2 and 8-3).<sup>3,79,156</sup>

Although children can sustain injuries almost anywhere, school and sports activities are the most prominent causes of soft tissue injuries occurring between the ages of 6 and 14 years old.<sup>12,155</sup>

Cuts, bruises, and abrasions make up almost three fourths of all injuries, and almost half of these injuries are in the face<sup>136,179</sup> (Figs. 8-4 through 8-6).

Dog bite injuries in young children (up to age 9) are another cause of soft tissue injuries where the face is most commonly damaged (Fig. 8-7).\*

Domestic violence (including child abuse) must be considered when we examine soft tissue injuries around the neck, head, mouth, and face, which are the most affected by physical (including intentional) injury.<sup>†</sup> Child abuse is a complex phenomenon in all countries.<sup>35,181</sup> Physicians and dentists who suspect child abuse must be prepared to assist these patients and take measures to help the victim prevent future assault.<sup>54,110,117</sup>

There are clear signs that show that a child has been abused such as:

1. The nature and location of the injury may be unusual for the age of the child.
2. The parents may be reluctant to describe how the injury occurred.
3. The description of the trauma may change each time the story is retold by the parents.
4. The parent may seem unconcerned, even when the child has obviously been injured.<sup>164</sup>

The most common injured sites in child abuse include the head, face, mouth, and neck.

The usual lesions are bruises, burns, welts, contusions, hematomas, ecchymosis especially in the face and eyes, scrapes, bone fractures, and dental injuries, which do not match the description of the trauma<sup>‡</sup> (Fig. 8-8).

Teenagers sustain injuries practicing sports without protective equipment. Adolescents often suffer severe injuries in the maxillofacial region from bicycle accidents, skateboard

\*References 16, 53, 80, 113, 146, 172.

†References 57, 82, 111, 115, 139, 177.

‡References 54, 57, 67, 82, 111, 115.





A



B



C

**Figure 8-2 A-C,** One-year-old girl fell from a baby chair and suffered avulsion of her maxillary right central incisor. **A,** She was referred to the office 1 day after the trauma. **B,** Note the gingival edema. **C,** The avulsed tooth.



**Figure 8-3** Three-year-old boy fell down in the bathroom, sustaining traumatic injuries in the lip and nose.



**Figure 8-4** A 10-year-old boy was hit with a hockey stick and suffered injuries to the soft and hard tissues.



**Figure 8-5** Fourteen-year-old boy was running and fell down against a hard floor. Profound bruise and scab over the upper lip.



**Figure 8-6** Eleven-year-old girl received a blow to the mouth with a baseball bat and sustained traumatic injuries to the soft and hard tissues.

falls, motorcycle crashes, etc.\* (Figs. 8-9 and 8-10). Studies of these traumatized patients have also shown injuries from equestrian sports; most common are head-first falls off a horse<sup>33,169,174</sup> (Fig. 8-11).

Another cause of facial trauma in adolescents is violence (mainly fights and assaults). Victims are predominantly males in their late teens and early twenties† (Fig. 8-12).

Adults sustain oral trauma most commonly from sporting activities, road traffic crashes, injuries at work, and physical assaults<sup>22,59,76</sup> (Fig. 8-13). Even the elderly are quite



**Figure 8-7** Eleven-year-old boy was attacked by a dog when he was 7 years old. Note the different scarring in his face.

susceptible to trauma (including oral trauma) due to visual deterioration, decline in cognitive ability, physical frailty, and/or loss of equilibrium<sup>101,127,166</sup> (Fig. 8-14).

## MANAGEMENT OF SOFT TISSUE INJURIES

Each type of soft tissue injury will be considered as a separate entity with its different traumatic lesions, their management, follow-up, and prevention.

## THE FACE

Facial trauma occurs most commonly following vehicle crashes. Facial trauma may be accompanied by deep wounds and jaw fractures<sup>1,13,72</sup> (see Chapter 7). Emergency treatment initially involves an assessment of the traumatic injury as described in Chapter 2, followed by any necessary procedures to control bleeding and to repair cuts, puncture wounds, lacerations, and teeth/jaw fractures. Additional procedures, subsequent to the initial repair, may include plastic surgery as a result of scars or deformities<sup>119,158</sup> (Fig. 8-15).

Wound closure using conventional sutures and/or cyanoacrylate plays an important role in wound repair. If this surgical procedure is performed adroitly, scars can be minimized.<sup>7,85,101</sup>

When properly performed, suturing will gently maintain the wound margins in their proper position, thus avoiding soft tissue ischemia, and allow healing by primary intention. There must be very close tissue adaptation with minimal

\*References 2, 29, 65, 68, 87, 90, 100, 101, 108.

†References 30, 55, 76, 93, 120, 131, 180.





A



B



C



D



E



F

**Figure 8-8** Six-year-old boy. The mother reported that he fell from a swing. **A**, Note the several injuries, including jaw fracture, a cut in the lower lip involving the orbicular muscle, and lateral luxation of the primary maxillary central incisors and both lateral incisors. Subluxation of mandibular permanent right central incisor and avulsion of the lateral incisor of the same side. Counter-blow hematomas and bruises in both cheeks. **B**, Profile view showing dislodgment of the jaw as consequence of the fracture. **C**, Biting on ice-cold gauze to stop the mucosal bleeding. **D**, Wound in the lower lip involving the orbicular muscle. **E**, Traumatic injury on the chin, the wound in the lower lip, and bruises on the face. **F**, A suture was placed along with cyanoacrylate.





**Figure 8-9** An 11-year-old girl fell off her bicycle, resulting in several bruises and hematomas.



A



B

**Figure 8-10** A 17-year-old female fell off her motorcycle. **A**, Sustaining hematoma, bruises, ecchymosis. **B**, Closer view of the contralateral side.



A



B



C

**Figure 8-11** A 17-year-old female was thrown from her horse. **A** and **B**, Photographs taken of the accident. **C**, Frontal view 2 days after the trauma. Note the bruises on the face and edema of upper lip. (**A** and **B**, Courtesy of Miss Sandra Azar, official photographer of the Argentine Rural Society.)



A



B

**Figure 8-12** Twenty-year-old female was a victim of domestic violence. **A**, Two days after the episode. She sustained blows to the zygoma; the hematoma is evident. **B**, A closer view.



**Figure 8-14** Eighty-one-year-old elderly women fell because of vertigo. The photograph shows several hematomas on the face, especially around the zygoma.



A



B

**Figure 8-13** A 25-year-old man 1 day after an automobile crash. **A**, Profile view showing nasal infraction. **B**, Clinical view of the mouth with several injuries to the soft and hard tissues.





A



B



C

**Figure 8-15** **A**, Same patient as Figure 8-1. Note that the patient needs additional plastic surgical procedures because of the deformities in the lower lip. **B**, Right profile of the same patient; her deformities are evident. **C**, Another view of the deformities in the lower lip and injuries in the hard tissue.

tension between the margins of the wound so that infection can be avoided. This enables rapid repair and minimizes scarring. In most cases scars result from the improper approximation of torn tissue and/or improper suturing<sup>40,86,175</sup> (Fig. 8-16, A and B).

Scars can remain unchanged for years and some scars will gradually fade over time. Scars can produce psychological impairments and social embarrassment for the patient, especially teenagers and adolescents.<sup>86,101,139,158</sup> Psychological problems are also common in adults as a consequence of disfiguring scars<sup>36,86,105</sup> (see Fig. 8-16, C and D).

When the face sustains injuries, especially to the chin, the clinician should suspect potentially associated hard tissue injuries (e.g., to the teeth and bone). The initial impact may be to the chin; however, this may lead to a secondary impact to the maxilla, known as a “counter-blow” (e.g., an *indirect impact*). Multiple fractures of the molars, intrusive luxation, and/or crown-root fractures are commonly found after chin trauma<sup>43,59,92,141,157</sup> (Figs. 8-17 to 8-19). Condylar head fractures are also a common injury following chin trauma. They are usually diagnosed by sudden malocclusion along with clinical signs such as an uncomfortable, painful, and limited mouth opening. Sometimes a condylar head fracture can be misdiagnosed,<sup>41,137</sup> resulting in severe functional consequences for the temporomandibular joint and leading to further temporomandibular joint disorder (TMD).<sup>42,92</sup> Chin injuries have a higher frequency in boys<sup>73</sup> (Figs. 8-20 and 8-21).

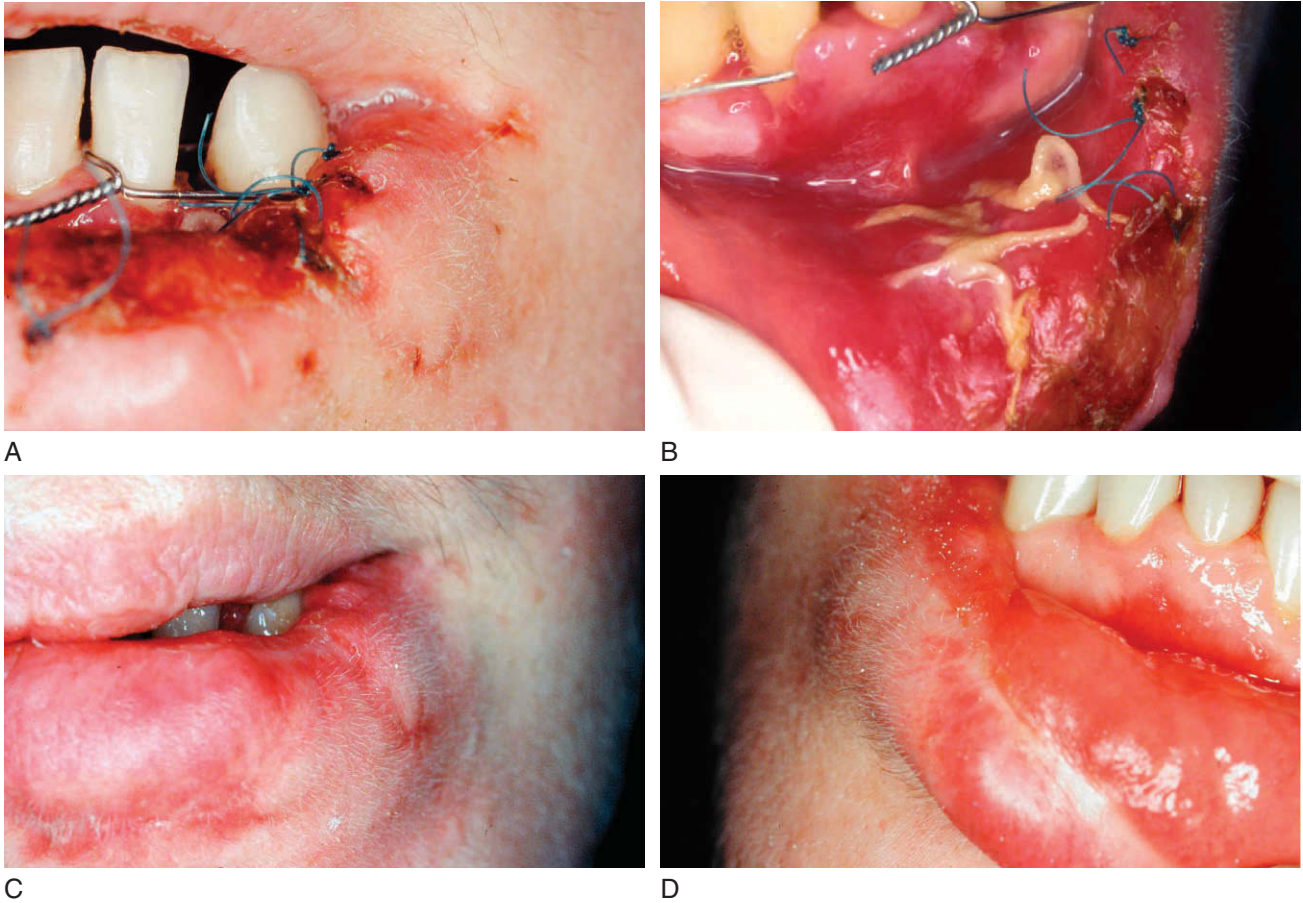
## IMMEDIATE TREATMENT OF FACIAL TRAUMA

If the injuries are minor, immediate treatment consists first of simply washing the patient’s face with sterile sponges or gauze soaked in an antiseptic soap until every visible particle has been removed. The clinician may find particles of teeth, wood, glass, dirt, etc. For this reason, it is very important to know where the trauma occurred. Cleaning the wounds initially and thoroughly in this manner substantially reduces bacterial contamination and wound infection,<sup>49,62,75,88,154</sup> and helps to visually determine the severity of the soft tissue injury (Fig. 8-22).

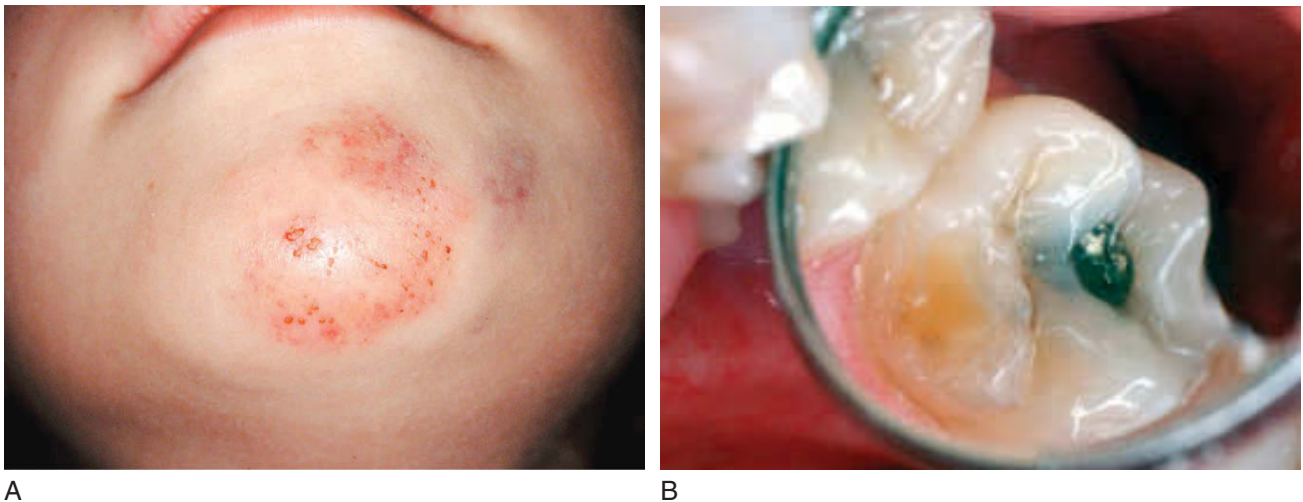
Foreign bodies deeply embedded in the patient’s face are sometimes very difficult to detect; retained, they can cause cosmetic disfigurement, tattooing, and scarring<sup>45,70,107</sup> (Fig. 8-23). These contaminated foreign bodies create a favorable environment for further infection.<sup>77,167</sup> Following facial cleaning and débridement, a tetanus vaccination is strongly recommended<sup>14,161</sup> along with broad-spectrum antibiotic therapy.\*

\*References 46, 49, 95, 97, 139, 173.





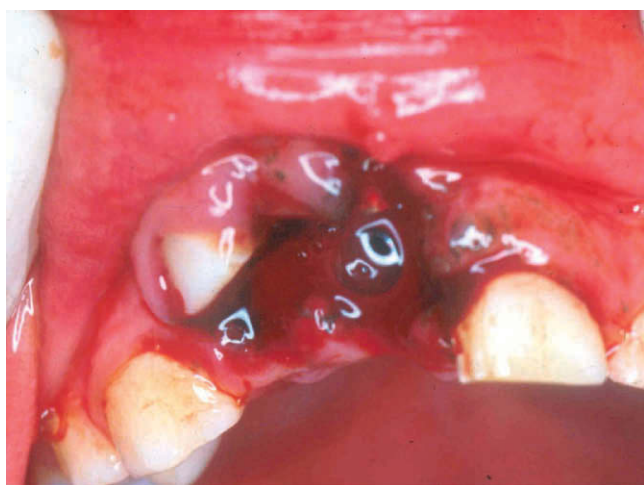
**Figure 8-16** **A**, An inappropriate lip suture was placed, causing loss of part of the orbicular muscle. Note the inappropriate splint attempting to stabilize the mandibular luxated teeth. **B**, Inappropriate suturing of the lip mucosa. **C**, One year later, the patient suffers with an avoidable disfigurement in the lower lip. **D**, Internal view of the lip. Note the scar in the mucosa.



**Figure 8-17** **A**, Eleven-year-old girl fell off her bicycle and hit her chin with a resultant hematoma and multiple ecchymoses. **B**, In the same patient a counter-blow to the first molar fractured off the palatal cusp.



A



B

**Figure 8-18** **A**, Three-year-old boy fell, hitting his chin with bruises and edema. **B**, The counter-blow caused severe intrusion of his primary left incisors. Note the deep wound in the gingiva and the coagulum.

## COMPLEMENTARY TREATMENT FOR FACIAL TRAUMA

Facial wound healing is a complex process with two factors important to consider:

- Endogenous factors (pathophysiological)
- Exogenous factors (microorganisms)<sup>25</sup>

To reduce the risk of facial infection after trauma, it is helpful to prescribe some complementary treatments such



**Figure 8-19** Three-year-old girl fell, hitting her chin. She sustained palatal luxation of both primary central incisors.



**Figure 8-20** A 10-year-old boy fell with a resulting hematoma on the chin.

as: topical antimicrobial agents and antimicrobial moisture dressings; even honey and essential oils have been recommended.<sup>25</sup>

Rose hip oil is one of the essential oils that the authors recommend. It is 100% pure, has no added chemicals, and has the effects of diminishing scars, (surgical or traumatic), fading color, and reducing of redness and hyperpigmentation. It may also decrease the formation of a keloid type of scar that may appear after surgical procedures. The rose hip oil is applied with only 2 or 3 drops onto the affected areas. It is placed at night, with a circular massage, until it is completely absorbed (usually within 2 to 3 minutes). The oil must be used daily for 2 months or more, depending on the magnitude of the scars. This treatment has been shown to be useful for the cosmetic improvement of scar appearance.<sup>124,160</sup>





**Figure 8-21** A 25-year-old male had a motorcycle crash. Note the deep cuts in the neck and the hematoma on the chin.

## THE LIPS

The lips are the most likely soft tissues to sustain injuries, occurring in approximately 85% of all facial injuries.<sup>5,8,40,66,138</sup> This is understandable since there would be a tendency for the lips to get “trapped” between two hard surfaces in the event of a traumatic injury, specifically between the teeth and the surface against which the lips sustain the impact. The direct impact of the lips during the trauma in conjunction with this compression injury causes the lips to suffer a double impact.<sup>40</sup>

Lip injuries can be internal and/or external. Lip wounds, including the orbicularis muscles, may involve simple lacerations, hematoma, or tooth fragment or foreign body embedment, which may not always be clinically visible (Figs. 8-24 through 8-30). Patients undergoing orthodontic treatment often suffer the most severe internal injuries<sup>169</sup> (Fig. 8-31).

Severe trauma (e.g., vehicular crashes) may lead to the loss of part of the lip muscular mass (see Figs. 8-15 and 8-16). These disfiguring oral injuries may have a significant effect on the patient’s functional, aesthetic, and psychological well being.<sup>40</sup>

The presence of foreign bodies embedded in the lips can cause infection and scarring; to prevent this, their early detection and surgical removal is essential.<sup>5,34,40,138,163</sup>

The recommended technique to remove intruded tooth fragments or foreign bodies is through an incision on the lip’s internal surface because it is easier and yields better healing results (Fig. 8-32, A and B). However, if the foreign body is visible on the lip’s external surface, it is more prudent to remove it through the external surface (see Fig. 8-30).

## CYANOACRYLATES AND THEIR DENTAL APPLICATIONS

In dentistry, cyanoacrylates have been used for many different purposes, including:

- as a tissue adhesive<sup>17,40</sup>
- in lieu of sutures<sup>17,18,47</sup>
- as a hemostatic agent<sup>4,19,20,39,64</sup>
- in lieu of calcium hydroxide for partial pulpotomy<sup>21,37</sup>
- for treating hypersensitive dentin<sup>81</sup>
- for treating aphthous ulcers<sup>91,151</sup>
- for preparing emergency splints<sup>109,144</sup>
- for periodontal surgery and apicoectomy for approximation of full thickness flaps<sup>64,69</sup>
- for sutureless closing of lip wounds and repair of minor injuries to the labial mucosa/frenum<sup>4,64</sup> thus reducing the risk of cheloid scarring<sup>40</sup> (see Fig. 8-38).

The repair of traumatic lip injuries and surgical incisions is described later in this chapter.\*

## CLINICAL EXAMINATION OF THE LIPS

The examination of the lips begins with careful visual observation using appropriate illumination and magnification. A comprehensive history about the circumstances of the trauma can be helpful in determining what kinds of foreign bodies might be found in the lips. Palpation of the lips is important to detect foreign bodies (Fig. 8-33).

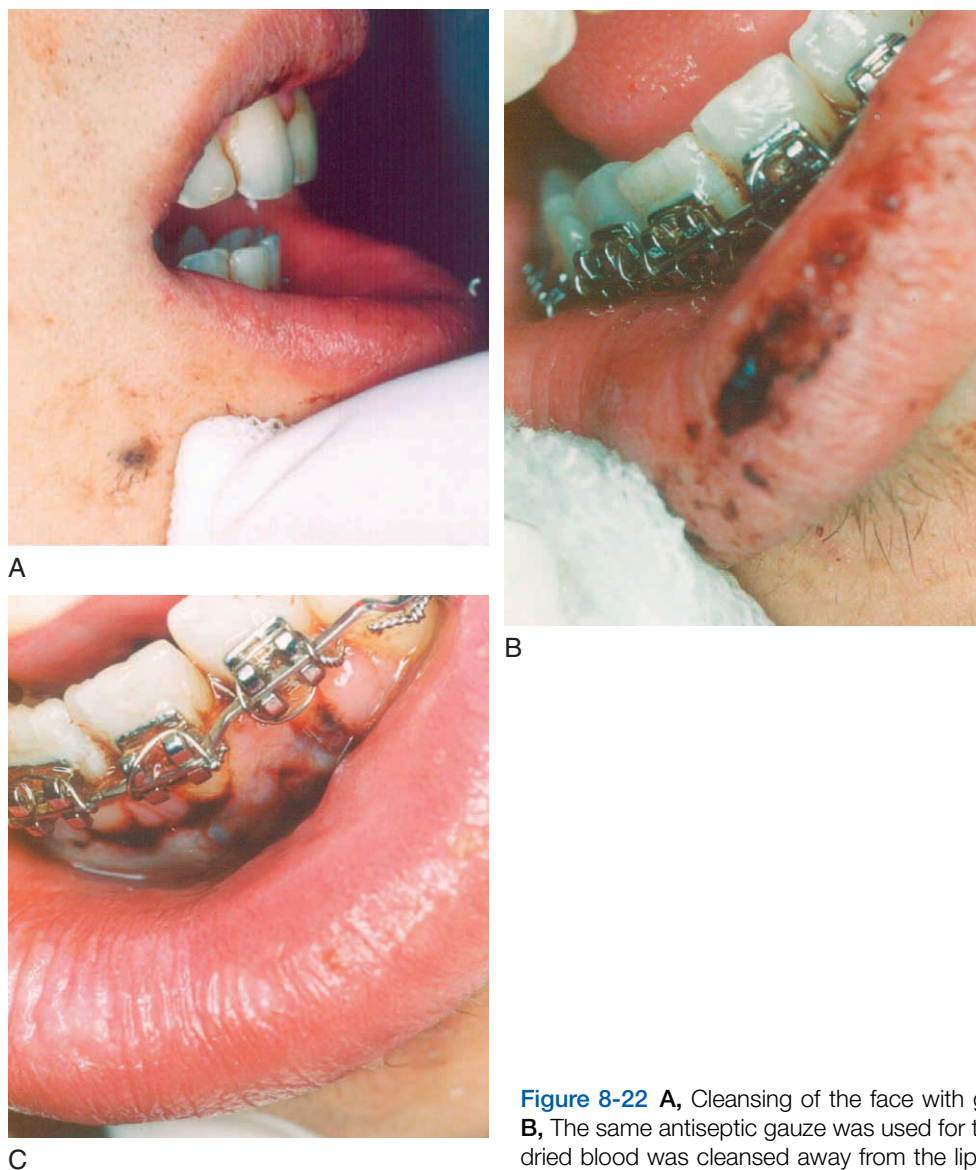
To further determine if unseen foreign bodies have been lodged in the lip(s), the clinician should gather answers to the following questions:

- Does the patient complain of pain in the lip(s)?
- Is (Are) the lip(s) edematous? (see Fig. 8-29, A-B)?
- Does palpation reveal any induration?
- Does palpation elicit pain?
- Is there missing tooth structure on any tooth adjacent to the injured lip(s)? (see Fig. 8-30)

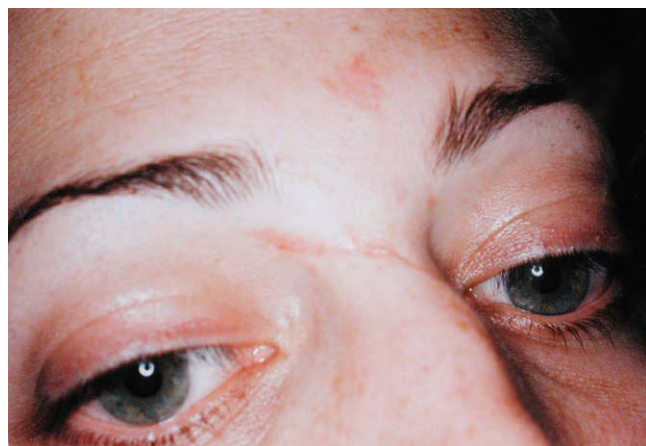
If answers to any of these questions are affirmative, then a radiographic examination of the lip(s) is recommended.

\*References 5, 8, 34, 40, 66, 103.

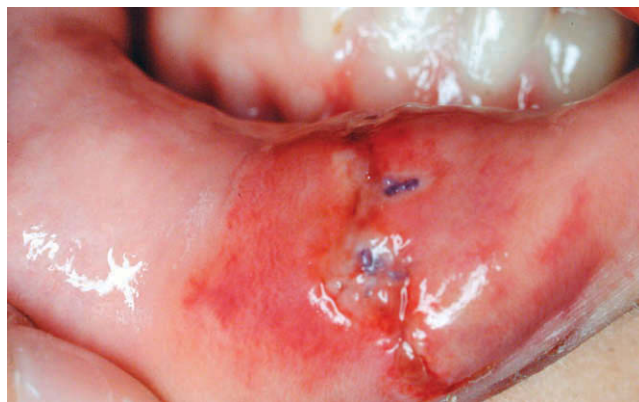




**Figure 8-22** A, Cleansing of the face with gauze soaked in an antiseptic soap. B, The same antiseptic gauze was used for the initial cleansing of the lips. C, The dried blood was cleansed away from the lips.



**Figure 8-23** A 24-year-old female, 1 year after automobile crash, facial particles of glass were embedded in her skin, leading to disfigurements.



**Figure 8-24** A 17-year-old female was hit in the mouth with a hockey puck, causing a deep internal wound in the lower lip with several hematomas.



A



B

**Figure 8-25** A, An 18-month-old girl fell out of a baby carriage, and presented with severely infected wound in the lower lip. B, Two days later the wound is repairing well.



**Figure 8-26** A 24-year-old man suffered a road traffic injury. He presented with several lesions in soft and hard tissues. The lip shows a deep wound with necrotic tissue, lacerations, and a hematoma.



**Figure 8-27** A 12-year-old boy fell, sustaining a wound in the lower lip with an infected vesicle.



A



B

**Figure 8-28** A, A 4-year-old girl fell, injuring her lip. She presented with an infected vesicle and hematoma. She had subluxation of both maxillary central incisors and a wound in the upper frenum. B, Radiograph of the lip showing several dental fragments embedded in the lip (arrows). The wound is also visible radiographically.

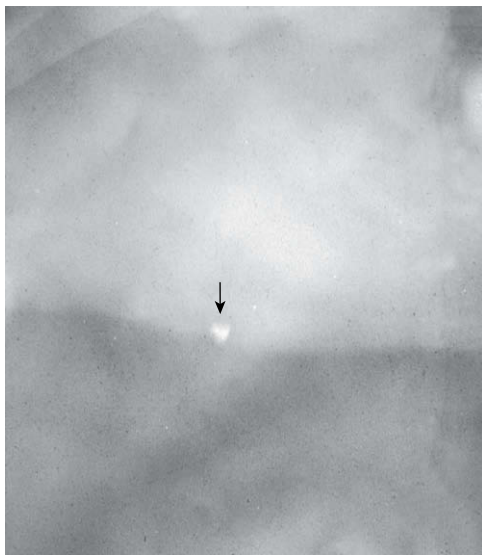




A

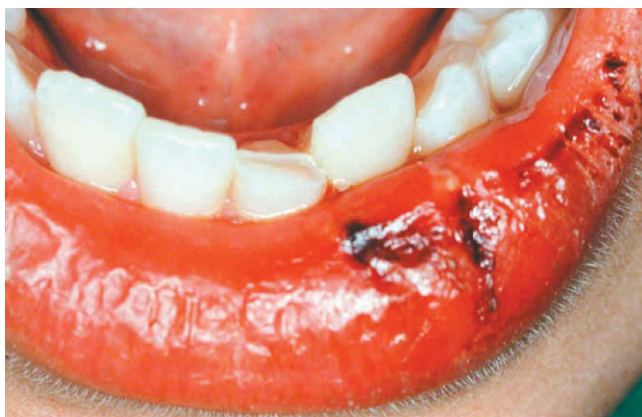


B



C

**Figure 8-29** A, A 17-year-old female was injured playing hockey. Note the edema of the right side, including the upper lip. She received previous medical assistance. B, Profile view. C, Radiograph of the upper lip showing tooth fragments embedded in the lip (*arrow*).

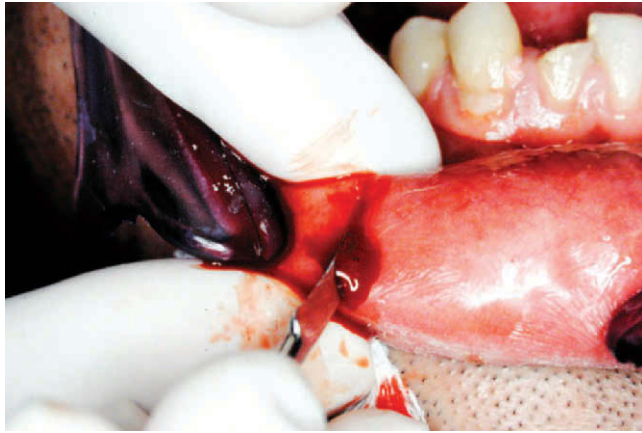


**Figure 8-30** A 12-year-old girl was hit with a hockey puck. She presented with injuries to the hard and soft tissues. In the lower lip, the tooth fragment is visible because it was forced just below the mucosa.



**Figure 8-31** An 18-year-old woman was undergoing orthodontic treatment as seen in this intraoral view of upper lip. She presents with a deep wound and traumatic ulcers produced by the brackets.





A



B

**Figure 8-32 A,** The recommended technique to remove embedded tooth fragments or foreign bodies is through an incision on the lip's internal surface because it is easier and yields better healing results. **B,** Using a small surgical spoon (Lucas spoon, Hufriedy N° CL 84) the tooth fragments are removed.

## RADIOGRAPHIC EXAMINATION OF THE LIPS

When a radiographic examination is necessary, the following technique is recommended:

- Place the film firmly in the vestibule facing the lip (Fig. 8-34).
- Reduce x-ray exposure time 50% (because it is only soft tissue); there is still a sufficient dosage of radiation to detect foreign bodies lodged in the lip.
- For horizontal and vertical reference points, use small strips of lead laminate from the film packet; one strip should be 3 mm in length, the other strip 6 mm in length. The lead strips should be taped on the small sides of the film (Fig. 8-35). As an alternative, steel paper clips can



A



B



C

**Figure 8-33 A,** Examination of the lips begins with careful visual observation with good illumination and appropriate magnification. **B,** The examination of the lip continues with careful palpation. The clinician is tactilely searching for induration indicating a foreign body is present; otherwise the lip feels uniformly soft. **C,** Another view of the palpation of the lip.



A



B

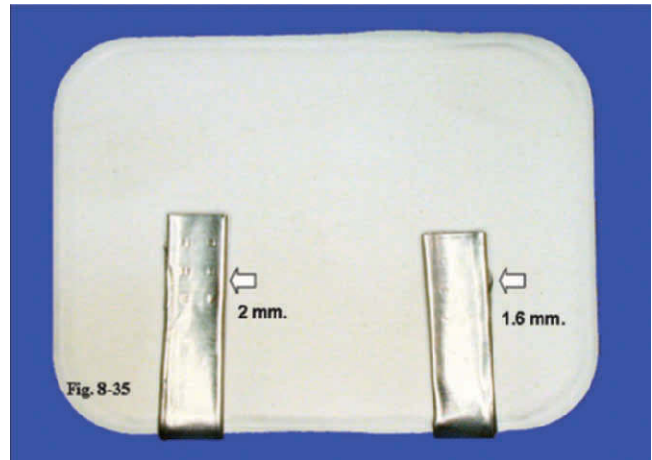
**Figure 8-34** **A**, The film is placed by the patient in the labial vestibule facing the lip. **B**, Another view.

also be used on this film to maintain orientation (see Fig. 8-38, *B*).

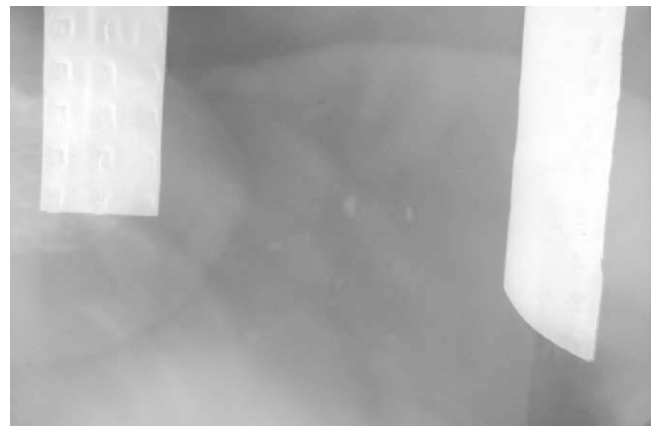
- This technique helps the practitioner orient left-from-right and top-from-bottom; however, the *depth* of the foreign body can only be determined by direct visual observation.

## TECHNIQUE FOR CLOSING LIP WOUND WITH CYANOACRYLATES

1. Flush the wound well with copious amounts of ice-cold sterile saline solution; the ice-cold stimulates vasoconstriction and provides excellent hemostasis.



**Figure 8-35** Film with vertical radiopaque reference points using small strips of metal laminate from the film packet.



**Figure 8-36** Radiograph showing several tooth fragments embedded in the lip. The reference points help the clinician approximate the location of these fragments.

2. Next, use sterile gauze soaked in ice-cold sterile saline, rubbing away all foreign bodies and dirt attached to the surface of the skin and lip.
3. If foreign bodies are still present, they must be surgically removed. Administer 2% lidocaine with 1:50,000 epinephrine (assuming there are no allergy or systemic contraindications) with a short 30-gauge needle. This will provide adequate anesthesia and prolonged hemostasis.
4. Confirm removal of all foreign bodies radiographically along with direct examination using good magnification (at least 4×) and fiberoptic illumination.
5. To maintain hemostasis, lightly squeeze the lip with the ice-cold sterile gauze. This pressure, maintained for several minutes, is quite effective for maximum hemostasis. This enables the clinician to confirm how the edges of the wound will approximate.



6. a. With the incision open, deposit a drop of cyanoacrylate in the deepest part of the wound.
- b. Reapproximate the wound edges, add another drop of cyanoacrylate on the edges of the wound, and maintain digital pressure for approximately 4 minutes.
- c. Remove excess cyanoacrylate with warm moist gauze or with scissors.

Occasionally, a single nylon suture may be necessary when the lip muscles are involved; this reduces the risk of the edges of the wound being pulled apart. If internal suturing is necessary, absorbable sutures are highly recommended.

Antibiotic therapy should be decided on a case-by-case basis. If the patient is healthy and the immune system is not compromised by systemic disease, current literature suggests that antibiotics are probably unnecessary.<sup>15,58,112</sup> If the patient

is systemically fragile with a compromised immune system or if there is clear evidence of an active infection (swelling, pain, purulence, etc.),<sup>11,31,118,132</sup> antibiotic therapy is probably a prudent approach.

When a resorbable suture has to be placed within the internal surface of the wound, the recovery process of the labial mucosa ranges from 1 to 2 weeks, depending on the depth of the wound (Figs. 8-37 through 8-39).

## ADVANTAGES OF CYANOACRYLATES

Many studies have shown the advantages of cyanoacrylates.

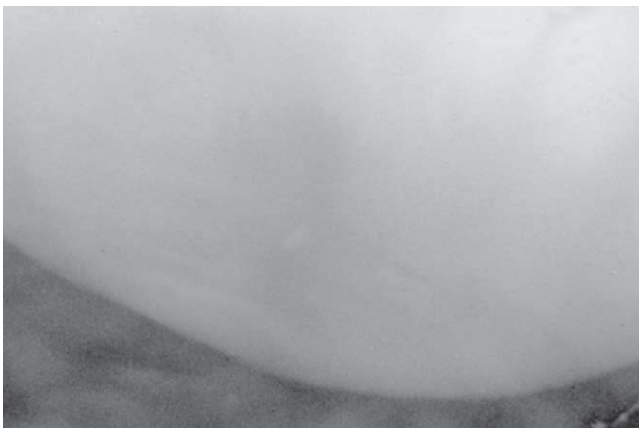
- They are excellent tissue adhesives.<sup>48,96,121,122</sup>
- Upon contacting soft tissue, polymerization begins immediately.<sup>48,50,106,122</sup>



A



B



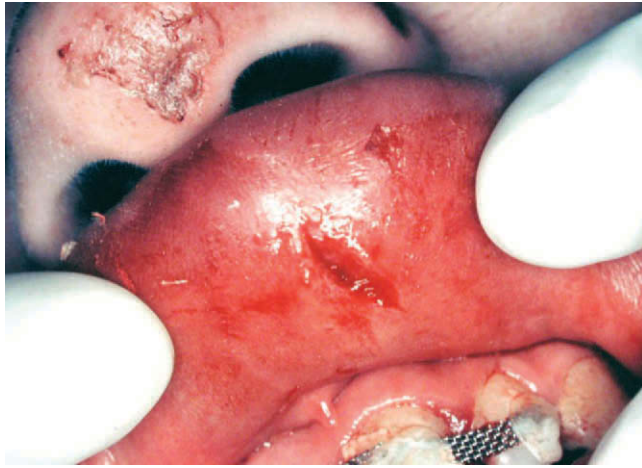
C



D

**Figure 8-37** **A**, A 14-year-old boy fell while running; 1 day after trauma. He received emergency medical care 1 day earlier. The upper lip is very edematous. He presented with lesions on the face along with several bruises. **B**, The bandage was removed. Note the inappropriate treatment. The suture was placed without cleaning and disinfection of the wound. **C**, Preoperative radiograph of the upper lip showing the embedded tooth fragment. **D**, The tooth fragment must be removed first to clean the lip. Particles of dirt forced into the wound during the trauma are attached to the skin. *Continued*





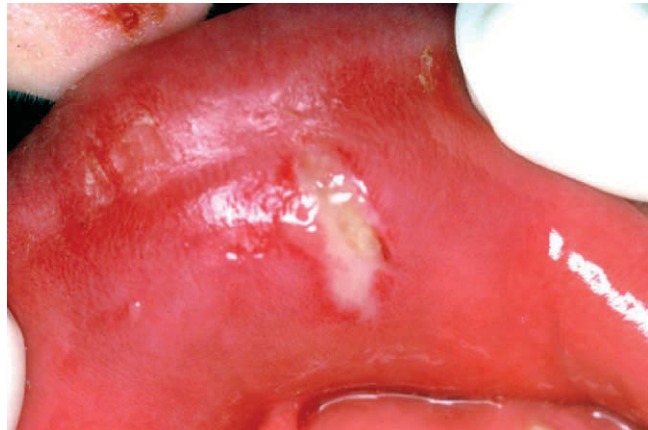
E



F



G



H



I

**Figure 8-37, cont'd** **E**, Incision to remove the embedded tooth fragment. **F**, Radiograph following extraction of the tooth fragment before suturing the wound closed. **G**, Closing the wound using only cyanoacrylate. Digital pressure over the wound site is maintained for approximately 4 minutes. **H**, Wound healing 2 days after closing with cyanoacrylate. **I**, Fifteen days later, the wound shows complete repair.

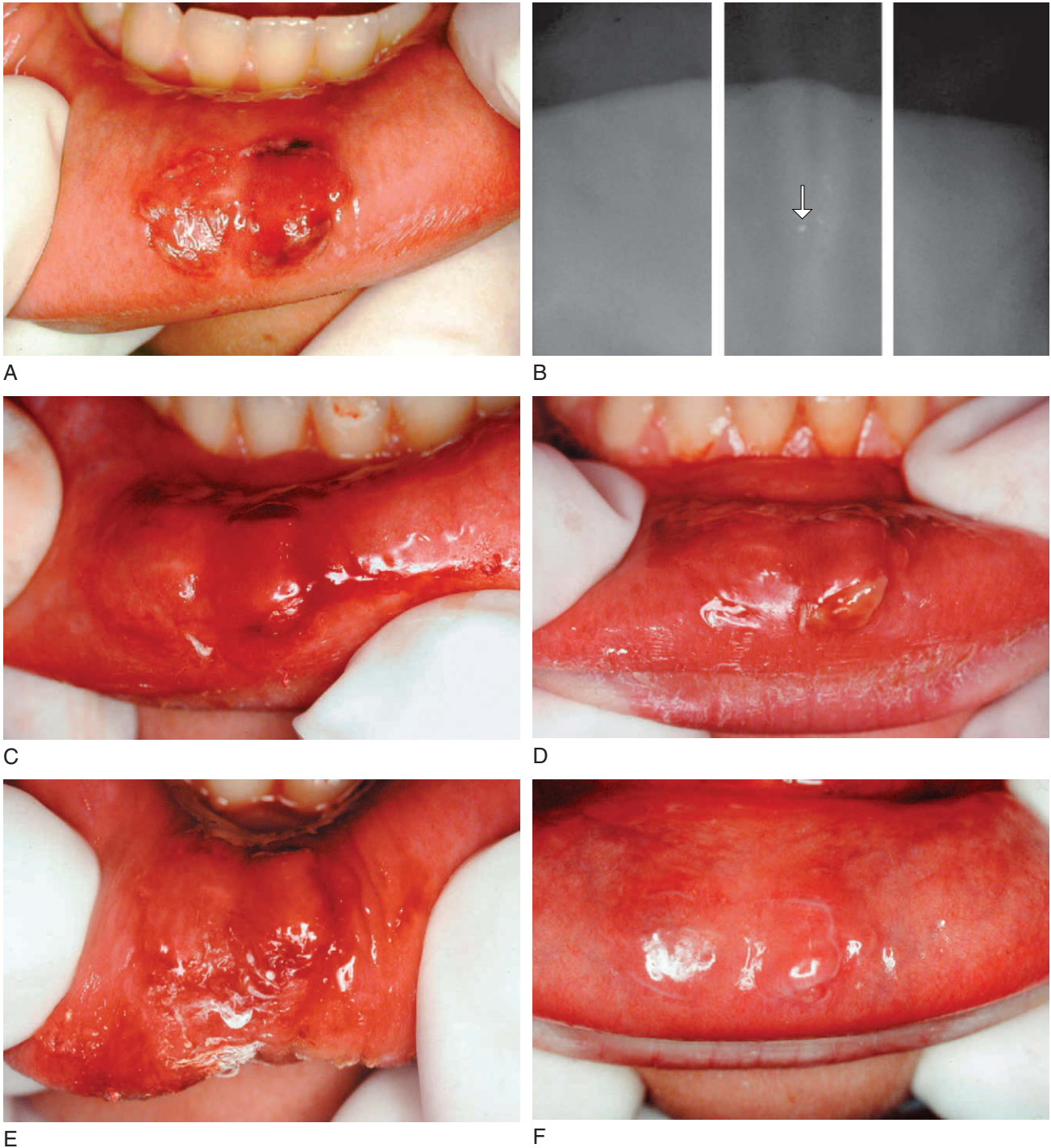
- They are biocompatible and hemostatic. Hemostasis takes place fast, which makes the surgical procedure much easier.\*
- They are effective against external bacterial invasion and contamination.†

\*References 4, 24, 39, 40, 48, 50, 64, 106, 122, 145, 154, 165.

†References 12, 24, 64, 112, 121, 133, 154, 165.

- Cyanoacrylates kill gram positive bacteria including *Staphylococcus*, *Pseudomonas*, and gram negative bacteria (e.g., *Escherichia coli*).\*
- Cyanoacrylates enable a quick, effective, painless, and needle-free method of closing a wound, thus assuring better compliance of patients and parents.<sup>74,84,135,153</sup>

\*References 52, 58, 64, 69, 112, 116, 154, 165.



**Figure 8-38** **A**, A 14-year-old girl fell, sustaining a deep cut in the lower lip. One day after trauma, the patient reported pain in her lip. **B**, Radiograph with reference points using a steel paper clip. The tooth fragments are embedded in the middle of the lip (*arrow*). **C**, After extraction of the tooth fragments and before approximating the wound edges using only cyanoacrylate. **D**, Another view of the wound before closing with cyanoacrylate. **E**, The wound closed with only cyanoacrylate. **F**, One week after closing with cyanoacrylate shows complete healing.





**Figure 8-39** **A**, A 10-year-old boy fell down and presented 2 hours after trauma. The deep wounds involved both orbicular muscles. Note the red abrasion on his cheek. The wounds were cleaned with sterile saline solution and antiseptic soap. **B**, Another view showing the deep wounds of his lips involving his orbicularis muscles and cheek abrasions. **C**, The wounds after closing with cyanoacrylate. **D**, Eighteen days following cyanoacrylate wound closure reveals good tissue repair with scabs covering the wounds. **E**, Six months later shows good repair with very little scarring. **F** and **G**, One year later, both wounds show complete healing with virtually no evidence of scarring.





G



H

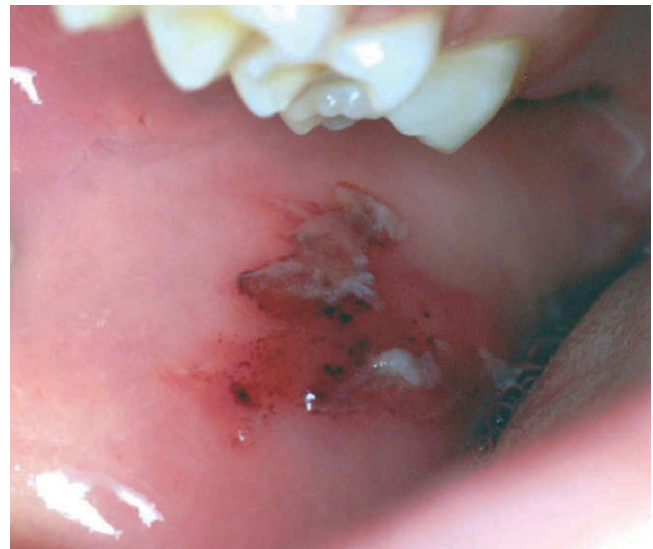
**Figure 8-39, cont'd H,** Twelve years later, an improved cosmetic appearance.

- The quickness of the surgical procedure improves the results.\*
- Cyanoacrylates are economic and provide a better cosmetic outcome.†

## DISADVANTAGES OF CYANOACRYLATES

Other studies have shown the disadvantages of cyanoacrylates, including:

- Cyanoacrylates cannot be used in wounds involving muscle attachments.<sup>50,62,106,128,147</sup>
- Cyanoacrylates are indicated only for topical or superficial wounds.<sup>26,145</sup>
- Cyanoacrylates are not advisable for sloughs, lacerations, bites, or nonsurgical puncture wounds.<sup>165</sup>
- Cyanoacrylates are contraindicated in lacerations of more than 5 cm.<sup>165</sup>
- Cyanoacrylates should not be used in contaminated wounds.<sup>165</sup>



**Figure 8-40** A 17-year-old female fell, injuring both cheeks. Note the lacerations and traumatic ulcers in the mucosa of the cheek.

## THE MUCOSA

Mucosal trauma includes the cheeks and periodontium.

- *Cheek injuries* are infrequent and often result from a “counter-blow” (e.g., trauma that secondarily causes the cheeks to be “trapped” between two hard surfaces, such as between the teeth of the maxillary and mandibular molars [Fig. 8-40]). Should fractured teeth occur from a traumatic injury to the face, the rough occlusal edges should be reduced so that the mucosa of the cheeks does not sustain additional trauma from tooth irritation.

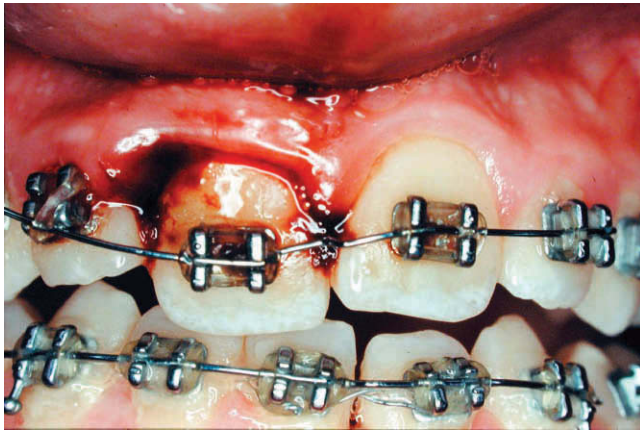
Traumatic ulcerations of the mucosa may result from continuous trauma from fractured teeth.

Some authors have suggested different treatments for these ulcerations such as:

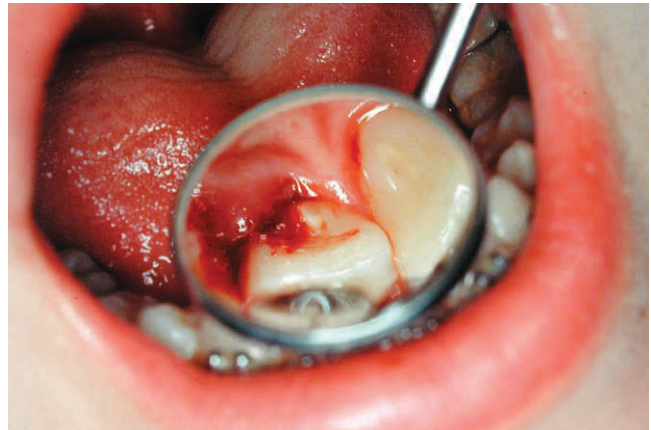
1. **Amlexanox** (Aphthasol™) 5% oral paste. This medication is an H-1 receptor antagonist like Claritin. It is essentially a nonsedating antihistamine and is the only FDA-approved drug specifically for this purpose. It is only mildly effective, but it is very useful in young patients if you do not want to use steroids.
2. **Orabase Sooth-N-Seal™** (OTC). This is a cyano-butyl-acrylate with benzocaine. It serves as a protectant and is considered biocompatible.

\*References 34, 40, 50, 84, 103, 106, 121, 122, 135, 142, 165.

†References 6, 26, 46, 75, 121, 122, 134, 145, 153.



A



B

**Figure 8-41** **A**, Eleven-year-old boy who suffered lateral luxation of the maxillary right central incisor and subluxation of the ipsilateral lateral incisor after a punch in the mouth. The patient presents gingival edema and sulcular bleeding, clear clinical signs that the PDL and alveolar bone are also affected by the trauma. Note the hematoma in the mucosa of the upper lip. **B**, Note the sulcular bleeding and edema in the palatal gingiva.

3. **Fluocinonide** cream, gel, or ointment 0.05%. Fluocinonide 0.05 (Lidex™) is a moderate potency topical steroid. Under no circumstances should Temovate (clobetasol) or Ultravate (halobetasol) be used as initial therapy for a mucosal ulceration. The absorption kinetics of these drugs is 5 to 7 times higher than Lidex and can result in the onset of cushingoid side effects within a 2-week period.<sup>151</sup>

■ *Injury to the periodontium* is an important clinical sign of trauma, specifically with dental luxation injuries. Sulcular bleeding indicates that the periodontal ligament (PDL) and alveolar bone have been affected by trauma<sup>28,32,143,171</sup> (Fig. 8-41). Avulsion, lateral, extrusive, and intrusive luxation often cause severe injuries to the mucosa, including deep wounds. These lacerations must be treated concurrently with hard tissue (teeth and alveolar bone) injuries<sup>28,143</sup> (Figs. 8-42 through 8-44).

**Initial treatment** for injuries to the mucosa consists of cleaning and pressing firmly on the affected area with chilled sterile saline solution to stop any bleeding.

**Intermediate treatment** includes prescribing a 0.12% chlorhexidine gel or an antiseptic mouthwash to reduce the oral flora in the affected area. The patient must clean the affected area 3 times per day for 1 week. If the patient is a child, parents must supervise this procedure<sup>62,159</sup> (Fig. 8-45).

Some serious traumatic injuries can produce a laceration in the marginal gingiva, causing the gingival tissue to become necrotic. In these cases, it is necessary to surgically remove the necrotic gingival pedicle under local anesthetic. Posttreatment care should include covering the gingiva with a palliative surgical cement dressing or cyanoacrylate for 1 or 2 days to allow for protected and uneventful tissue healing (Fig. 8-46).<sup>40</sup>

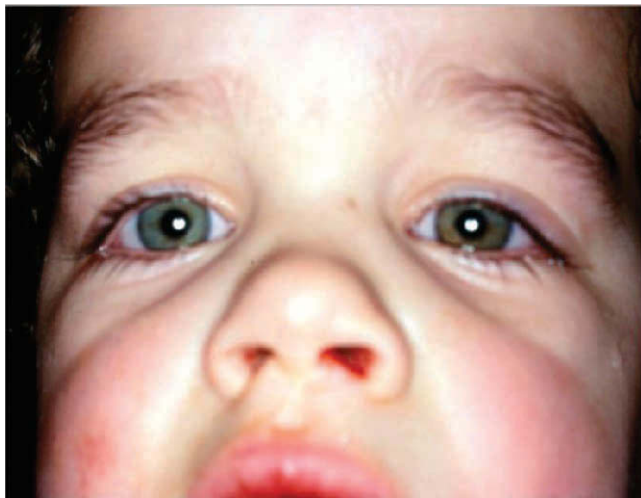


**Figure 8-42** A 23-year-old man was assaulted. He was referred to the dental office 3 days after the assault with avulsion of the maxillary left central incisor, subluxation of the maxillary left lateral incisor, lateral luxation of the maxillary right central incisor, and subluxation of the ipsilateral lateral incisor.

## THE LABIAL FRENUM

Labial frenum injury is common in very young children (between 1 and 4 years old). Often when these toddlers fall, they do so with their mouths open and they may fall against a hard object (Figs. 8-47 and 8-48). The emergency treatment consists of careful flushing and cleaning of the wound with copious amounts of ice-cold sterile saline solution for hemostasis. An oral antiseptic mouthrinse (e.g., 0.12% chlorhexidine solution) should be used to flush the wound two or three times per day. In most of the cases, the lesion repairs by itself and suturing is usually not necessary (Fig. 8-49). If the wound is deep, a single inter-





A

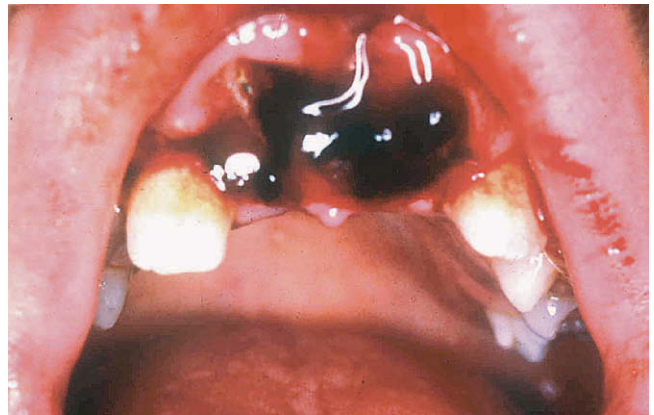


B

**Figure 8-43** **A**, A 2-year-old boy 2 days after trauma. The bloody nose was caused by the same trauma. **B**, He sustained severe intrusion of the maxillary left central incisor, hematoma in the labial frenum attachment, and trauma to the periodontium and sulcus.



A



B

**Figure 8-44** **A**, A 12-year-old female 5 days after trauma. She sustained a serious counter-blow to the chin and lower lip. **B**, She sustained severe intrusion of the maxillary central incisors as reflected by continuous bleeding through the gingival wound characteristic of intrusion injuries. **C**, Preoperative radiograph showing the severe intrusion of both incisors. The left incisor has a midroot fracture; the right incisor shows significant displacement along with rotation.



C

*Continued*



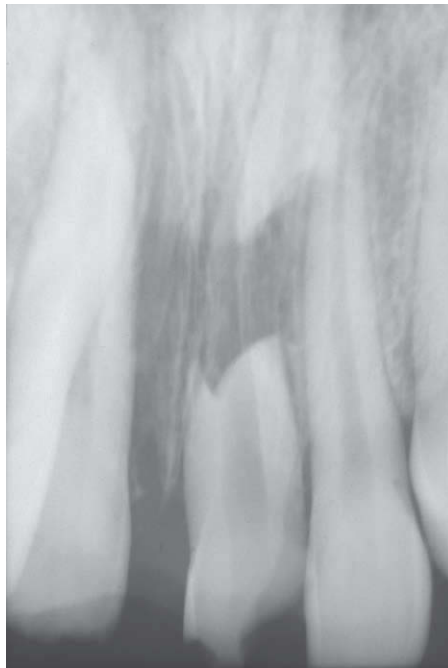


D

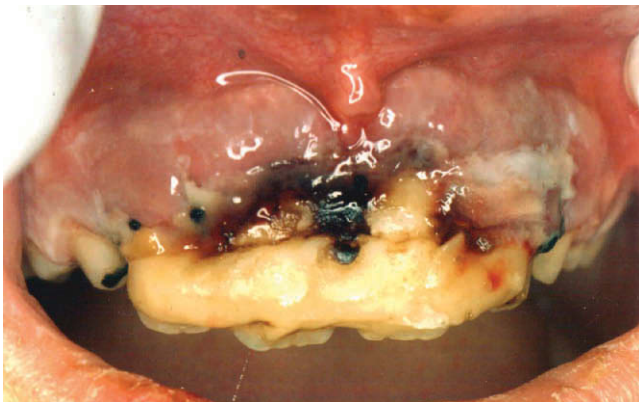
**Figure 8-44, cont'd D,** The intruded teeth after their surgical reposition. Note the soft tissue was repositioned and sutured in place. **E and F,** Radiographs exposed after surgical repositioning. Note the major displacement of the apical fragment of the root.



E



F



A



B

**Figure 8-45 A and B,** Eight-year-old boy, who was referred 3 days after immediate replantation of avulsed maxillary incisors following a car crash. The teeth were replanted in the hospital 1 hour after avulsion. Note the poorly constructed splint and the plaque on the mucosa.

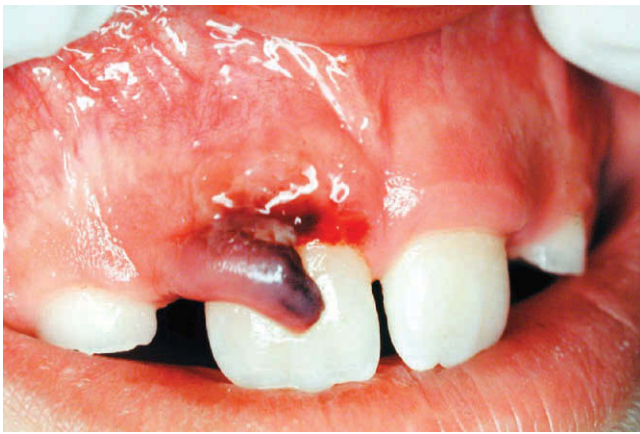


C



D

**Figure 8-45, cont'd** C, The gingival flap was surgically removed. D, An appropriate splint was constructed 3 days later. This splinting included primary canines. Note the rapid gingival healing as a result of the chlorhexidine rinsing.



A



B



C

**Figure 8-46** A, Eight-year-old boy 4 hours after being hit in the mouth with a ball, resulting in concussion to the maxillary right central incisor and a necrotic pedicle laceration of the gingiva. B, Surgical removal of the necrotic pedicle gingival tissue. C, The wound is covered with a surgical cement dressing.





**Figure 8-47** An 18-month-old girl fell, causing subluxation of the maxillary right central and lateral incisors with a deep wound that involved the labial frenum and surrounding mucosa.



**Figure 8-48** A 2½-year-old girl fell, causing subluxation of both maxillary incisors and a deep wound in the labial frenum and mucosa.



**A**



**B**

**Figure 8-49** **A**, A 3-year-old boy fell, causing a deep wound in the labial frenum with copious bleeding. **B**, One week later rapid healing is evident. The parents washed the affected area three times per day with chlorhexidine.

rupted suture may be indicated along with cyanoacrylates<sup>4,34,64,69</sup> (Fig. 8-50).

## THE TONGUE

Tongue injury has the same etiology as a cheek injury in that its occurrence is infrequent and usually results from a “counter-blow,” especially when the patient falls down and bites his or her tongue. Because the tongue has a rich vascular supply, the deeper the wound, the greater the number of layers to suture.<sup>10</sup> Antibiotic therapy is usually recommended.<sup>177</sup>

In general, tongue injuries usually occur on the lateral borders, but occasionally the injury may occur in the midline<sup>66,168</sup> (Fig. 8-51).

## HEALING MECHANISM OF ORAL SOFT TISSUE WOUNDS

Wound healing is a complex physical and chemical process<sup>78,88,129</sup> that can be separated into 4 different phases<sup>94,178</sup>:





A



B



C

**Figure 8-50** **A**, An 8-year-old girl hit her open mouth against the metal border of a placard, causing a deep wound in the labial frenum and significant bleeding. **B**, Wound closure with cyanoacrylate. **C**, Four days later, showing removal of the suture and cyanoacrylate. Note how quickly the wound is repairing.



**Figure 8-51** A 28-year-old woman fell down 20 years ago, causing her incisors to penetrate her tongue. Note the persistent scarring.

1. Hemostasis
2. Inflammatory response
3. Proliferation
4. Remodeling<sup>44,78,178</sup>

According to Diegelmann<sup>44</sup>: “The normal healing response begins the moment the tissue is injured. As the blood components spill into the site of injury, the platelets come into contact with exposed collagen and other elements of the extracellular matrix. This contact triggers the platelets to release clotting factors and essential growth factors and cytokines, such as platelet derived growth factor (PDGF) and transforming growth factor beta (TGF- $\beta$ ). Following hemostasis, the neutrophils enter the wound site and begin the critical task of phagocytosis to remove foreign body materials, bacteria, and damaged tissue. As part of the inflammatory phase, macrophages also appear, releasing more PDGF and TGF- $\beta$ . Once the wound site is cleaned out, the fibroblasts migrate in to begin the proliferative phase and deposition of the new extracellular matrix. The new collagen matrix becomes cross-linked and organized during the final remodeling phase.”<sup>44</sup>

Prostaglandin E2 (PGE2) has been reported to control angiogenesis and play an important role in wound healing in soft tissue.<sup>140</sup> Since basic fibroblast growth factor (bFGF) has been reported to generate neovascularization, PGE2 and bFGF might work closely or one might control the expression of the other.<sup>88,178</sup>

Wound healing could be affected by:

- **Local factors:** e.g., edema, ischemia, low oxygen tension,<sup>60,162</sup> and infection.
- **Regional factors:** e.g., arterial and venous insufficiency and neuropathy.
- **Systemic factors:** e.g., inadequate perfusion and metabolic disease.
- **Miscellaneous factors:** e.g., nutritional state, preexisting illness, history of radiation therapy, and smoking.<sup>78</sup>

According to Phillips<sup>129</sup>: “The optimal rate of healing is approached when factors advantageous to healing are present and factors having the ability to disturb or retard the healing processes are controlled or absent.”

## PREVENTIVE MEASURES TO AVOID SOFT TISSUE INJURIES

As further described in Chapter 11, there are measures that can be taken to decrease the possibility of traumatic injuries to intraoral and extraoral soft tissue. But to summarize:

- Make sure that children wear appropriate protective gear, such as hard shell and foam helmets, to prevent head and facial injuries while bicycle riding, roller blading, skateboarding, etc.<sup>28,89,164</sup>
- Encourage the use of custom-fabricated mouthguards to prevent tooth-related injuries during contact sports.<sup>29,68,99,102,125</sup>
- Use appropriate vehicle safety restraints (seat belts) and appropriately sized car seats for children.\* Seat belts significantly reduce the risk of maxillofacial skeletal injuries; injuries to the skull, brain, and spinal cord; and the incidence of concussions, contusions, and cerebral lacerations.<sup>51,98,150</sup> Furthermore, passengers without seat belts may also sustain thoracic and abdominal trauma and limb injuries. These traumatic injuries may produce severe disability or even death.<sup>9,71,126,148</sup>
- Airbags are another important preventive measure and have been shown to dramatically reduce the severity of automobile injuries.<sup>1,72,98,104,123</sup>

## CONCLUSION

The main objectives in this chapter were to educate dentists on how to treat, manage, and prevent soft tissue injuries, thus avoiding posttraumatic sequelae such as scarring,

tattoos, or other healing defects. Following the protocols described in this chapter will enable the clinician to minimize the unfavorable esthetic, functional, and psychological sequelae that accompany improper treatment.

## REFERENCES

1. Abbas I, Ali K, Mirza YB: Spectrum of mandibular fractures at a tertiary care dental hospital in Lahore, *J Ayub Med Coll Abbottabad* 15:12-14, 2003.
2. Acton CH, Nixon JW, Clark RC: Bicycle riding and oral/maxillofacial trauma in young children, *Med J Aust* 165:249-251, 1996, *Comment Int Med J Aust* 5:166:510, 1997.
3. Agran PF, Anderson C, Winn D et al: Rates of pediatric injuries by 3-month intervals for children 0 to 3 years of age, *Pediatrics* 111(6 Pt 1):e683-692, 2003.
4. Al-Belasy FA, Amer MZ: Hemostatic effect of n-butyl-2-cyanoacrylate (histoacryl) glue in warfarin-treated patients undergoing oral surgery, *J Oral Maxillofac Surg* 61:1405-1409, 2003.
5. Allen FJ: Dental fragments in the lips, *Int J Oral Surg* 10(Suppl 1):260-263, 1981.
6. Applebaum JS, Zalut T, Applebaum D: The use of tissue adhesion for traumatic laceration repair in the emergency department, *Ann Emerg Med* 22:1190-1192, 1993.
7. Armstrong BD: Lacerations of the mouth, *Emerg Med Clin North* 18:471-480, 2000.
8. Atiyeh BS, Ioannovich J, Al-Amm CA et al: Improving scar quality: a prospective clinical study, *Aesthetic Plast Surg* 26:470-476, 2002.
9. Baker DR, Clarke SR, Brandt EN: An analysis of factors associated with seat belt use: prevention opportunities for the medical community, *J Okla State Med Assoc* 93:496-500, 2000.
10. Bankole OO, Fasola AO, Denloye OO: Oro-facial soft tissue injuries in Nigerian children: a five-year review, *Afr J Med Sci* 33:93-97, 2004.
11. Basilio RC, Loducca FE, Haddad PC: Medical dental prophylaxis of endocarditis, *Braz J Infect Dis* 8:340-347, 2004, *Epub* 7, 2005.
12. Bastone EB, Freer TJ, McNamara JR: Epidemiology of dental trauma: a review of the literature, *Aust Dent J* 45:2-9, 2004.
13. Batstone M, Monsour F, Pattel P: Transfer of facially injured road trauma victims and its impact on treatment, *ANZ J Surg* 75:411-414, 2005.
14. Becking AG: Tetanus prophylaxis in injuries. Guidelines for the dental practice, *Ned Tijdschr Tandheelkd* 102:266-268, 1995.
15. Bernard L, Doyle J, Friedlander SF et al: A prospective comparison of octyl cyanoacrylate tissue adhesive (derma-bond) and suture for the closure of excisional wounds in children and adolescents, *Arch Dermatol* 137:1177-1180, 2001.
16. Bernardo LM, Gardner MJ, Rosenfield RL et al: A comparison of dog bite injuries in younger and older children treated in a pediatric emergency department, *Pediatr Emerg Care* 18:247-249, 2002.
17. Bhaskar SN, Frisch J: Use of cyanoacrylate adhesive dentistry, *J Am Dent Assoc* 77:831-876, 1968.
18. Bhaskar SN: Tissue adhesives in dentistry. A review, *J Can Dent Assoc* 38:337-343, 1972.
19. Bhaskar SN, Frisch J, Cutright DE et al: Effect of butyl cyanoacrylate on the healing of extraction wounds, *Oral Surg Oral Med Oral Pathol* 24:604-616, 1967.

\*References 104, 177, 150, 126, 152, 164.

20. Bhaskar J, Colond DC: Tissue response of rat tongue to normal and isobutylcyanoacrylate, *Oral Surg Oral Med Oral Pathol* 26:573-578, 1968.
21. Bhaskar SN, Beasley JD, Ward JP et al: Human pulp capping with isobutyl cyanoacrylate, *J Dent Res* 51:58-61, 1972.
22. Bishai D, McCauley J, Trifiletti LB et al: The burden of injury in preschool children in an urban Medicaid managed care organization, *Ambul Pediatr* 2:279-283, 2002.
23. Blinkhorn FA: The aetiology of dento-alveolar injuries and factors influencing attendance for emergency care of adolescents in the north west of England, *Endod Dent Traumatol* 16:162-165, 2000.
24. Blondeel PN, Murphy JW, Debrosse D et al: Closure of long surgical incisions with a new formulation of 2-octylcyanoacrylate tissue adhesive versus commercially available methods, *Am J Surg* 188:307-313, 2004.
25. Bowler PG: Wound pathophysiology, infection and therapeutic options, *Ann Med* 34:419-427, 2002.
26. Bruns TB, Simon HK, McLario DJ et al: Laceration repair using a tissue adhesive in a children's emergency department, *Pediatrics* 98(4 Pt 1):673-675, 1996.
27. Bulinski P, Bachulis B, Naylor DF Jr et al: The changing face of trauma management and its impact on surgical resident training, *J Trauma* 54:161-163, 2003.
28. Chan AW, Heung GS, Ho MW: Different treatment outcomes of two intruded permanent incisors—a case report, *Dent Traumatol* 17:275-280, 2001.
29. Chapman HR, Curran AL: Bicycle helmets—does the dental profession have a role in promoting their use? *Br Dent J* 196:555-560, disc 539, 2004.
30. Cheng TL, Schwarz D, Brenner RA et al: Adolescent assault injury: risk and protective factors and locations of contact for intervention, *Pediatrics* 112:931-938, 2003.
31. Cohen S, Burns RC: Endodontic pharmacology. In Cohen S, Burns R, editors: *Pathways of the pulp*, ed 8, St Louis, 2002, Mosby.
32. Cohen S, Burns RC: Traumatic injuries. In Cohen S, Burns R, editors: *Pathways of the pulp*, ed 8, St Louis, 2002, Mosby.
33. Costa Paz M, Aponte-Tinao L, Muscolo DL: Injuries to polo riders: a prospective evaluation, *Br J Sports Med* 33:329-331, 1999.
34. Coulthard P, Worthington H, Esposito M et al: Tissue adhesives for closure of surgical incisions, *Cochrane Database Syst Rev* (2):CD004287, 2004. *Cochrane Database Syst Rev* (3):CD004287, 2003.
35. Coulthard P, Yong S, Adamson L et al: Domestic violence screening and intervention programmes for adults with dental or facial injury, *Cochrane Database Syst Rev* (2):CD004486, 2004.
36. Cusack KJ, Frueh BC, Brady KT: Trauma history screening in a community mental health center, *Psychiatr Serv* 55:157-162, 2004.
37. Cvek M, Granath L, Cleaton-Jones P et al: Hard tissue barrier formation in pulp-tomized monkey teeth capped with cyanoacrylate or calcium hydroxide for 10 and 60 minutes, *J Dent Res* 66:611-617, 1987.
38. da Silva AC, Passeri LA, Mazzonetto R et al: Incidence of dental trauma associated with facial trauma in Brazil: a 1-year evaluation, *Dent Traumatol* 20:6-11, 2004.
39. Davis SC, Eaglstein WH, Cazzaniga AL et al: An octyl-2-cyanoacrylate formulation speeds healing of partial-thickness wounds, *Dermatol Surg* 27:783-788, 2001.
40. de Blanco L: Lip suture with isobutyl cyanoacrylate, *Endod Dent Traumatol* 10:15-18, 1994.
41. Defabianis P: TMJ fractures in children: clinical management and follow-up, *J Clin Pediatr Dent* 25:203-208, 2001.
42. Defabianis P: Posttraumatic TMJ internal derangement: impact on facial growth (findings in a pediatric age group), *J Clin Pediatr Dent* 27:297-303, 2003.
43. Diab M, el Badrawy HE: Intrusion injuries of primary incisors, part I: review and management, *Quintessence Int* 31:327-334, 2000.
44. Diegelmann RF, Evans MC: Wound healing: an overview of acute, fibrotic and delayed healing, *Front Biosci* 9:283-289, 2004.
45. Duskova M: Treatment of traumatic tattooing of the face, *Rozhl Chir* 68:165-168, 1989.
46. Edwards R, Harding KG: Bacteria and wound healing, *Curr Opin Infect Disc* 17:91-96, 2004.
47. Eklund MK, Kent JN: The use of isobutyl cyanoacrylate as post-extraction dressing in humans, *J Oral Surg* 32:264-268, 1974.
48. Elmasalme FN, Matbouli SA, Zuberi MS: Use of tissue adhesive in the closure of small incisions and lacerations, *Pediatr Surg* 30:837-838, 1995.
49. Eriksson E, Vranckx J: Wet wound healing: from laboratory to patients to gene therapy, *Am J Surg* 188(1A Suppl):36-41, 2004.
50. Farion K, Osmond MH, Hartling L et al: Tissue adhesives for traumatic lacerations in children and adults, *Cochrane Database Syst Rev* (3):CD003326, 2002.
51. Farrell TM, Sutton JE, Clark DE et al: Moose-motor vehicle collisions. An increasing hazard in northern New England, *Arch Surg* 131:377-381, 1996.
52. FDA approves use of Dermabond adhesive to seal out infection-causing bacteria. First wound-closure. Technology approved to protect wounds and incisions from common microbes, Somerville, NJ, 2002, Johnson & Johnson press release.
53. Feldman KA, Trent R, Jay MT: Epidemiology of hospitalizations resulting from dog bites in California, 1991-1998, *Am J Public Health* 94:1940-1941, 2004.
54. Fenton SJ, Bouquot JE, Unkel JH: Orofacial considerations for pediatric, adult, and elderly victims of abuse, *Emerg Med Clin North Am* 18:601-617, 2000.
55. Froberg U, Bonsmann M: Orofacial injuries in skateboard accidents, *Aktuelle Traumatol* 22:84-86, 1992.
56. Gassner R, Bösch R, Tuli T et al: Prevalence of dental trauma in 6000 patients with facial injuries: implications for prevention, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 87:27-33, 1999.
57. Geddes JF, Whitwell HL: Inflicted head injury in infants, *Forensic Sci Int* 16 146:83-88, 2004.
58. Giray CB, Us D, Guney C et al: Antibacterial and cytotoxic effects of N-butyl-2-cyanoacrylate used as a tissue adhesive, *Mikrobiyol Bul* 27:154-163, 1993.
59. Glendor U, Halling A, Bodin L et al: Direct and indirect time spent on care of dental trauma: a 2-year prospective study of children and adolescents, *Endod Dent Traumatol* 16:16-23, 2000.
60. Gordillo GM, Sen CK: Revisiting the essential role of oxygen in wound healing, *Am J Surg* 186:259-263, 2003.
61. Gore GC, Magdalinos H, Pless IB: School injuries and preventive policies and programs, *Can J Public Health* 95:424-428, 2004.
62. Gouin S, Patel H: Office management of minor wounds, *Can Fam Physician* 47:769-774, 2001.
63. Greene D, Raven R, Carvalho G et al: Epidemiology of facial injury in blunt assault. Determinants of incidence and outcome in 802 patients, *Arch Otolaryngol Head Neck Surg* 123:923-928, 1997.
64. Grisdale J: The use of cyanoacrylates in periodontal therapy, *J Can Dent Assoc* 64:623-633, 1998.



65. Grossman DC: The history of injury control and the epidemiology of child and adolescent injuries, *Future Child* 10:23-52, 2000.
66. Hague BA, Honnas CM: Traumatic dental disease and soft tissue injuries of the oral cavity, *Vet Clin North Am Equine Pract* 14:333-347, 1998.
67. Hahn YS, Raimondi AJ, McLone DG et al: Traumatic mechanisms of head injury in child abuse, *Childs Brain* 10:229-241, 1983.
68. Hansen KS, Engesaeter LB, Viste A: Protective effect of different types of bicycle helmets, *Traffic Inj Prev* 4:285-290, 2003.
69. Herod EL: Cyanoacrylates in dentistry: a review of the literature, *J Can Dent Assoc* 56:331-334, 1990.
70. Herrmann A, Wohlrab J, Marsch WC: Trauma due to blank cartridges and fireworks. Early and correct treatment prevents tattooing and scarring, *Unfallchirurg* 107:22-26, 2004.
71. Hertz ES: A note on the variance of paired comparisons estimates, *Accid Anal Prev* 34:401-404, 2002.
72. Hogg NJ, Stewart TC, Armstrong JE et al: Epidemiology of maxillofacial injuries at trauma hospitals in Ontario, Canada, between 1992 and 1997, *J Trauma* 49:425-432, 2000.
73. Holan G: Traumatic injuries to the chin: a survey in a paediatric dental practice, *Int J Paediatr Dent* 8:143-148, 1998.
74. Holger JS, Wandersee SC, Hale DB: Cosmetic outcomes of facial lacerations repaired with tissue-adhesive, absorbable, and nonabsorbable sutures, *Am J Emerg Med* 22:254-257, 2004.
75. Hollander JE, Singer AJ, Valentine SM et al: Risk factors for infection in patients with traumatic lacerations, *Acad Emerg Med* 8:716-720, 2001.
76. Howe A, Crilly M: Violence in the community: a health service view from a UK, *Accident and Emer Department Public Health* 116:15-21, 2002.
77. Hung YT, Hung LK, Griffith JF et al: Ultrasound for the detection of vegetative foreign body in hand—a case report, *Hand Surg* 9:83-87, 2004.
78. Hunt TK, Hopf H, Hussain Z: Physiology of wound healing, *Adv Skin Wound Care* 13(2 Suppl):6-11, 2000.
79. Ivanenko YP, Dominici N, Cappellini G et al: Kinematics in newly walking toddlers does not depend upon postural stability, *J Neurophysiol* 94:754-763, 2005.
80. Javaid M, Feldberg L, Gipson M: Primary repair of dog bites to the face: 40 cases, *J R Soc Med* 91:414-416, 1998.
81. Javid B, Barkhordar RA, Bhinda SV: Cyanoacrylate—a new treatment for hypersensitive dentin and cementum, *J Am Dent Assoc* 114:486-488, 1987.
82. Jessee SA: Physical manifestations of child abuse to the head, face and mouth: a hospital survey, *ASDC J Dent Child* 62:245-249, 1995.
83. Junkins EP, Knight S, Olson LM et al: Analysis of school injuries resulting in emergency department or hospital admission, *Acad Emerg Med* 8:343-348, 2001.
84. Karcioğlu O, Göktas N, Coskun F et al: Comparison of tissue adhesive and suturing in the repair of lacerations in the emergency department, *Eur J Emerg Med* 9:155-158, 2002.
85. Keast DH, Bowering CK, Evans AW et al: MEASURE: A proposed assessment framework for developing best practice recommendations for wound assessment, *Wound Repair Regen* 12(3 Suppl):S1-17, 2004.
86. Key SJ, Thomas DW, Shepherd JP: The management of soft tissue facial wounds, *Br J Oral Maxillofac Surg* 33:76-85, 1995.
87. Kligman MD, Knotts FB, Buderer NM et al: Railway train versus motor vehicle collisions: a comparative study of injury severity and patterns, *J Trauma* 47:928-931, 1999.
88. Komarcevic A: The modern approach to wound treatment, *Med Pregl* 53:363-368, 2000.
89. Kubiak R, Slongo T: Unpowered scooter injuries in children, *Acta Paediatr* 92:50-54, 2003.
90. Kumamoto DP, Maeda Y: A literature review of sports-related orofacial trauma, *Gen Dent* 52:270-280; quiz 281, 2004.
91. Kutcher MJ, Ludlow JB, Samuelson AD et al: Evaluation of a bio-adhesive device for the management of aphthous ulcers, *Am Dent Assoc* 132:368-376, 2001.
92. Kvaal SI, Kvaal B: Tooth and jaw injuries following violence—diagnosis and treatment in emergency department, *Tidsskr Nor Lægeforen* 10:120:843-847, 2000.
93. Laski R, Ziccardi VB, Broder HL et al: Facial trauma: a recurrent disease? The potential role of disease prevention, *J Oral Maxillofac Surg* 62:685-688, 2004.
94. Lawrence WT: Physiology of the acute wound, *Clin Plast Surg* 25:321-340, 1998.
95. Legent F, Arnould JF: Antibiotic prophylaxis in ORL surgery and oral medicine, *Ann Fr Anesth Reanim* 13(5 Suppl):S100-199, 1994.
96. Lemaire D, Mongeau J, Dorion D: Microvascular anastomosis using histoacryl glue and an intravascular soluble stent, *J Otolaryngol* 29:199-205, 2000.
97. LeRoy JL Jr, Rees TD, Nolan WB: Infections requiring hospital readmission following face lift surgery: incidence, treatment, and sequelae, *Plast Reconstr Surg* 93:533-536, 1994.
98. Lestina DC, Williams AF, Lund AK: Motor vehicle crash injury patterns and the Virginia seat belt law, *JAMA* 265:1409-1413, 1991.
99. Lin MR, Hwang HF, Kuo NW: Crash severity, injury patterns, and helmet use in adolescent motorcycle riders, *J Trauma* 50:24-30, 2001.
100. Linn S, Smith D, Sheps S: Epidemiology of bicycle injury, head injury, and helmet use among children in British Columbia: a five year descriptive study. Canadian Hospitals Injury, Reporting and Prevention, *Inj Prev* 4:122-125, 1998.
101. MacAfee KA: Primary care management of maxillofacial injuries in sports, *Penn Dent J* 93:16-17, 25, 1994.
102. Macpherson AK, To TM, Macarthur C et al: Impact of mandatory helmet legislation on bicycle-related head injuries in children: a population-based study, *Pediatrics* 110(5):e60, 2002.
103. Magee WP, Ajkay N, Githae B et al: Use of octyl-2-cyanoacrylate in cleft lip repair, *Ann Plast Surg* 50:1-5, 2003.
104. Major MS, MacGregor A, Bumpous JM: Patterns of maxillofacial injuries as a function of automobile restraint use, *Laryngoscope* 110:608-611, 2000.
105. Matthew ML: Relationship between compensation claims for psychiatric injury and severity of physical injuries from motor vehicle accidents, *MJA* 175:129-132, 2001.
106. Mattick A: Use of tissue adhesives in the management of paediatric lacerations, *Med J* 19:382-385, 2002.
107. Matton G: Soft tissue trauma of the face, *Acta Chir Belg* 91:192-197, 1991.
108. Matz SO, Nibbelink G: Injuries in intercollegiate women's lacrosse, *Am J Sports Med* 32:608-611, 2004.
109. McCabe MJ: Use of histoacryl adhesive to manage an avulsed tooth, *BMJ* 301:20, 1990.
110. McDowell JD: Forensic dentistry. Recognizing the signs and symptoms of domestic violence: a guide for dentists, *J Okla Dent Assoc Fall* 88:21-28, 1997.
111. McMahon P, Grossman W, Gaffney M et al: Soft-tissue injury as an indication of child abuse, *J Bone Joint Surg Am* 77:1179-1183, 1995.
112. Mertz PM, Davis SC, Cazzaniga AL et al: Barrier and antibacterial properties of 2-octyl cyanoacrylate-derived wound treatment films, *J Cutan Med Surg* 71-76, 2003. *Epub* 09, 2002.
113. Mitchell RB, Nanez G, Wagner JD et al: Dog bites of the scalp, face, and neck in children, *Laryngoscope* 113:492-495, 2003.

114. Morad M, Vardi G, Kandel I et al: Trends in adolescent injury mortality in Israel, *Int J Adolesc Med Health* 16:279-283, 2004.
115. Naidoo S: A profile of the oro-facial injuries in child physical abuse at a children's hospital, *Child Abuse Negl* 24:521-534, 2000.
116. Narang U, Mainwaring L, Spath G et al: In-vitro analysis for microbial barrier properties of 2-octyl cyanoacrylate-derived wound treatment films, *J Cutan Med Surg* 7:13-19, 2003. *Epub* 27, 2002.
117. Needleman HL: Child abuse and neglect—recognition and reporting, *J Am Coll Dent* 61:30-37, 1994.
118. Oliver R, Roberts GJ, Hooper L: Penicillins for the prophylaxis of bacterial endocarditis in dentistry, *Cochrane Database Syst Rev* (2):CD003813, 2004.
119. Omovie EE, Sherperd JP: Assessment of repair of facial lacerations, *Br J Oral Maxillofac Surg* 35:237-240, 1997.
120. Ong TK, Dudley M: Craniofacial trauma presenting at an adult accident and emergency department with an emphasis on soft tissue injuries, *Injury* 30:357-363, 1999.
121. Osmond MH, Quinn JV, Sutcliffe T et al: A randomized, clinical trial comparing butylcyanoacrylate with octylcyanoacrylate in the management of selected pediatric facial lacerations, *Acad Emerg Med* 6:171-177, 1999.
122. Osmond MH, Klassen TP, Quinn JV: Economic comparison of a tissue adhesive and suturing in the repair of pediatric facial lacerations, *J Pediatr* 126:892-895, 1995.
123. Orsay EM, Dunne M, Turnbull TL et al: Prospective study of the effect of safety belts in motor vehicle crashes, *Ann Emerg Med* 19:258-261, 1990.
124. Ozcan M: Nutrient composition of rose (*Rosa canina* L.) seed and oils, *J Med Food* 5:137-140, 2002.
125. Padilla R, Dorney B, Balikov S: Prevention of oral injuries, *J Calif Dent Assoc* 24:30-36, 1996.
126. Passman C, McGwin G Jr, Taylor AJ: Seat belt use before and after motor vehicle trauma, *J Trauma* 51:105-109, 2001.
127. Peel NM, Kassulke DJ, McClure RJ: Population based study of hospitalised fall related injuries in older people, *Inj Prev* 8:280-283, 2002.
128. Penoff J: Skin closures using cyanoacrylate tissue adhesives. Plastic Surgery Educational Foundation DATA Committee. Device and Technique Assessment, *Plast Reconstr Surg* 103:730-731, 1999.
129. Phillips SJ: Physiology of wound healing and surgical wound care, *ASAIO J* 46:S2-5, 2000.
130. Powell EC, Jovtis E, Tanz RR: Incidence and description of high chair-related injuries to children, *Ambul Pediatr* 2:276-278, 2002.
131. Pratt HD, Greydanus DE: Adolescent violence: concepts for a new millennium, *Adolescent Med* 11:103-125, 2000.
132. Prieto-Prieto J, Calvo A: Microbiological basis of oral infections and sensitivity to antibiotics, *Med Oral Patol Oral Cir Bucal* 9 Suppl 15-18; 11-14, 2004.
133. Quinn JV, Osmond MH, Yurack JA et al: N-2-butylcyanoacrylate: risk of bacterial contamination with an appraisal of its antimicrobial effects, *J Emerg Med* 13:581-585, 1995.
134. Quinn JV, Drzewiecki A, Li MM et al: A randomized, controlled trial comparing a tissue adhesive with suturing in the repair of pediatric facial lacerations, *Ann Emerg Med* 22:1130-1135, 1993.
135. Quinn JV, Wells G, Sutcliffe T et al: A randomized trial comparing octylcyanoacrylate tissue adhesive and sutures in the management of lacerations, *JAMA* 277:1527-1530, 1997.
136. Ramirez M, Peek-Asa C, Kraus JF: Disability and risk of school related injury, *Inj Prev* 10:21-26, 2004.
137. Regev E, Zeltser R, Shteyer A: The overlooked chin trauma in children, *Refuat Hapeh Vehashinayim* 19:56-61, 79, 2002.
138. Rhee ST, Colville C, Buchman SR: Conservative management of large avulsions of the lip and local landmarks, *Pediatr Emerg Care* 20:40-42, 2004.
139. Rothman DL: Pediatric orofacial injuries, *J Calif Dent Assoc* 4:37-42, 1996.
140. Sakai Y, Fujita K, Sakai H et al: Prostaglandin E2 regulates the expression of basic fibroblast growth factor messenger RNA in normal human fibroblasts, *Kobe J Med Sci* 47:35-45, 2001.
141. Sasaki H, Ogawa T, Kawaguchi M et al: Multiple fractures of primary molars caused by injuries to the chin: report of two cases, *Endod Dent Traumatol* 16:43-46, 2000.
142. Saxena AK, Willital GH: Octylcyanoacrylate tissue adhesive in the repair of pediatric extremity lacerations, *Am Surg* 65:470-472, 1999.
143. Schatz JP, Joho JB, Dietschi D: Treatment of luxation traumatic injuries: definition and classification in the literature, *Pract Periodontics Aesthet Dent* 12:781-786, quiz 788, 2000.
144. Scheer B: Emergency treatment of avulsed teeth, *BMJ* 301:4, 1990.
145. Schwade ND: Wound Adhesives, 2-Octyl Cyanoacrylate, last updated, April 10, 2002.
146. Shaikh ZS, Worrall SF: Epidemiology of facial trauma in a sample of patients aged 1-18 years, *Injury* 33:669-671, 2002.
147. Shapiro AJ, Dinsmore RC, North JH: Tensile strength of wound closure with cyanoacrylate glue, *Am Surg* 67:1113-1135, 2001.
148. Shimamura M, Yamazaki M, Fujita G: Method to evaluate the effect of safety belt use by rear seat passengers on the injury severity of front seat occupants, *Accid Anal Prev* 37(1):5-17, 2005.
149. Shulman JD, Peterson J: The association between incisor trauma and occlusal characteristics in individuals 8-50 years of age, *Dent Traumatol* 20:67-74, 2004.
150. Shults RA, Elder RW, Sleet DA et al: Primary enforcement seat belt laws are effective even in the face of rising belt use rates, *Accid Anal Prev* 36:491-493, 2004.
151. Siegel MA, Silveman SS, Sollecito TP: editors: *Clinicians guide to common oral lesions*, ed 6, Burlington, Ontario, 2005, BC Decker, Inc.
152. Siegmund GP, Chimich DD, Heinrichs BE et al: Variations in occupant response with seat belt slack and anchor location during moderate frontal impacts, *Traffic Inj Prev* 6:38-43, 2005.
153. Simon HK, McLario DJ, Bruns TB et al: Long-term appearance of lacerations repaired using a tissue adhesive, *Pediatrics* 99:193-195, 1997.
154. Singer AJ, Quinn JV, Thode HC Jr et al: Trauma seal study group. Determinants of poor outcome after laceration and surgical incision repair, *Plast Reconstr Surg* 110:429-435; disc 436-437, 2002.
155. Skaare AB, Jacobsen I: Dental injuries in Norwegians aged 7-18 years, *Dent Traumatol* 19:67-71, 2003.
156. Smith GA, Bowman MJ, Luria JW et al: Babywalker-related injuries continue despite warning labels and public education, *Pediatrics* 100:E1, 1997.
157. Soviero VM, Guimaraes L, Miasato JM et al: Traumatic fractures of primary molars: a case report, *Int J Paediatr Dent* 7:255-258, 1997.
158. Spauwen PH: Soft tissue injuries of the face, *Ned Tijdschr Tandheelkd* 104:421-424, 1997.
159. Sreenivasan PK, Mattai J, Nabi N et al: A simple approach to examine early oral microbial biofilm formation and the effects of treatments, *Oral Microbiol Immunol* 19:297-302, 2004.

160. Szentmihalyi K, Vinkler P, Lakatos B et al: Rose hip (*Rosa canina* L.) oil obtained from waste hip seeds by different extraction methods, *Bioresour Technol* 82:195-201, 2002.
161. Talan DA, Abrahamian FM, Moran GJ et al: Tetanus immunity and physician compliance with tetanus prophylaxis practices among emergency department patients presenting with wounds, *Ann Emerg Med* 43:305-314, 2004, *Comment in: Ann Emerg Med* 43:315-317, 2004.
162. Tandara AA, Mustoe TA: Oxygen in wound healing—more than a nutrient, *World J Surg* 28:294-300, 2004, *Epub Feb*, 2004.
163. Taran A, Har-Shai Y, Ullmann Y: Traumatic self-inflicted bite with embedded tooth fragments in the lower lip, *Ann Plast Surg* 2:431-433, 1994.
164. The Merck Manual of Medical Information, Edit Pocket Books: pp: 1442, 172-172-169-Thomas B. Bruns TB, Mack Worthington J: Using tissue adhesive for wound repair: a practical guide to Dermabond, *Am Fam Physician* 61:1383-1388, 1997.
165. Thomas B, Bruns B, Mack Worthington J: Using tissue adhesive for wound repair: A practical guide to dermabond, *Am Fam Physician* 61:1383-1388, 1997.
166. Tornetta P III, Mostafavi H, Riina J et al: Morbidity and mortality in elderly trauma patients, *J Trauma* 46:702-706, 1999.
167. Toy BR, Frank PJ: Outbreak of *Mycobacterium* abscesses infection after soft tissue augmentation, *Dermatol Surg* 29:971-973, 2003.
168. Toure S, Fall I, Diallo BK: Emergency reimplantation of the tongue after complete traumatic amputation, *Rev Stomatol Chir Maxillofac* 104:52-54, 2003.
169. Travess H, Roberts HD, Sandy J: Orthodontics. Part 6: Risks in orthodontic treatment, *Br Dent J* 196:71-77, 2004.
170. Ueek BA, Dierks EJ, Homer LD, Potter B: Patterns of maxillofacial injuries related to interaction with horses, *Horse Oral Maxillofac Surg* 62:693-696, 2004.
171. Vinckier F, Declerck D, Verhaeghe V et al: Injuries to the permanent teeth. Periodontal lesions, *Rev Belge Med Dent* 53:105-151, 1998.
172. Walton RL, Beahm EK, Brown RE et al: Microsurgical replantation of the lip: multi-institutional experience, *Past Reconstr Surg* 102:8-68, 1998.
173. White RJ, Cooper R, Kingsley A: Wound colonization and infection: the role of topical antimicrobials, *Br J Nur* 10: 563-578, 2001.
174. Whitlock MR: Injuries to riders in the cross country phase eventing: the importance of protective equipment, *B J Sports Med* 33:212-214, 1999.
175. Widgerow AD, Chait LA, Stals R et al: New innovations in scar management, *Aesthetic Plast Surg* 24:227-234, 2000.
176. Wilson S, Smith GA, Preisch J et al: Epidemiology of dental trauma treated in an urban pediatric emergency department, *Pediatr Emerg Care* 13:12-15, 1997.
177. Wolfe Comer R, Gordon Fitchie J, Caughman FW et al: Emergency care of lacerations, fractures, and burns, *Oral Trauma* 85:34-41, 1989.
178. Wong ME, Hollinger JO, Pinero GJ: Integrated processes responsible for soft tissue healing, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 82:475-492, 1996.
179. Wood EB, Freer TJ: A survey of dental and oral trauma in south-east Queensland during 1998, *Aust Dent J* 47:142-146, 2002.
180. Zerfowski M, Bremerich A: Facial trauma in children and adolescents, *Clin Oral Investig* 2:120-124, 1998.
181. Zink T, Kamine D, Musk L et al: What are providers' reporting requirements for children who witness domestic violence? *Clin Pediatr* 43:449-460, 2004.



# THE LAW AND DENTAL TRAUMA

■ ■ ■  
ARTHUR CURLEY

## CHAPTER OUTLINE

### DENTAL LAWS AND LICENSING REGULATIONS

#### LEGAL ISSUES

- Standards of Care
- Unprofessional Conduct
- Violations of Statutes

#### CONSENT ISSUES

- Informed Consent/Refusal
  - Informed Consent
  - Informed Refusal

#### BURDEN OF PROOF

#### CAUSATION

#### CONDUCT OF OTHERS

#### GOOD FAITH SETTLEMENT

#### NATURE OF INJURY

#### NATURE OF THE TRAUMA

#### STANDARDS FOR TRAUMA CASES

#### GOOD SAMARITAN DEFENSES

#### NONINTENTIONAL INJURY/ACCIDENT/SPORTING CASES

#### ABUSE AND BATTERY CASES

- Confidentiality and Privileges
- Immunity
- Failure to Report
- Recognition of Abuse
  - Child Abuse
  - Abuse of the Elderly or Disabled

#### IATROGENIC TRAUMA CASES

#### CONCLUSION

Trauma patients present some unique legal issues for the modern dental practitioner as compared with long-term or regularly scheduled patients. At the same time, fundamental legal principles apply to all patients regardless of the origin or nature of the dental pathological condition presented. Therefore the prudent dental care provider should have a working knowledge of current dental law and the special issues presented in cases of trauma. This chapter will review issues of the standard of care, exceptions to that standard, record keeping requirements, communication and confidentiality of patient information, the doctrines of informed consent and informed refusal, referral duties, abuse reporting duties, and obligations regarding iatrogenic trauma patients.

Unlike human anatomy and physiology, some aspects of dental law can vary from state to state. Therefore this chapter can only offer general principles and will endeavor to point out areas where the practitioner would be well advised to consult local resources, such as dental societies, professional liability carriers, state licensing agencies, or attorneys with expertise in dental law in any particular state.

## DENTAL LAWS AND LICENSING REGULATIONS

The practice of dentistry is regulated by the laws of each state wherein the treatment is performed and the dental health care provider is licensed. Those laws generally regulate the practice of dentistry by setting forth required conduct, prohibited conduct, scope of practice, and regulation of licensed and nonlicensed support staff.

In addition, the practice of dentistry can also be regulated by laws found in states' Criminal, Insurance, Civil, and Health & Safety Code:

[http://www.dbc.ca.gov/b\\_and\\_p\\_code.htm](http://www.dbc.ca.gov/b_and_p_code.htm).

## LEGAL ISSUES

A dental health care provider can be subject to a claim or suit for malpractice if it can be proven there was negligent treatment that legally caused injury to a patient. When all three are present—*negligence*, *causing* (known as causation), and *injury*—a patient is entitled to payment for damages suffered.

*Malpractice* is known in the law as health care negligence, as the result of substandard care, unprofessional conduct, or violation of a statute:

<http://www.medicalmalpractice.com/dental-malpractice.cfm>.

## STANDARDS OF CARE

The *standard of care* is the most common measure of dental negligence. It is typically described as what a reasonable and prudent practitioner would do, or should have done, or should have avoided doing in the same or similar circumstances, in the same or similar locality, and during the same period of time. State to state variation of this doctrine is minimal. The source of any particular standard of care, with some exceptions, are not found in any legal treaty, statute, or text. Rather, it is determined by the statements or testimony of expert witnesses, who review records and statements or testimony in a case, in which they render opinions on the standard of care. A typical malpractice claim is determined by the *finder of fact*, a jury or judge sitting in place of a jury, determining which side's experts are to be believed. Such experts typically will support their opinions by citing texts, studies, journals, and personal experience:

<http://www.dentalbeacon.org/para3.htm>.

Until recently, the standard of care evolved slowly because of the legal qualifier of *a same or similar* locality during the same time period, sometimes called the *local standard of care qualifier*. In the past, it was understood that a rural dentist might not be expected to be as up-to-date on studies and other developments in dentistry as someone working in a metropolitan area with ready access to literature and colleagues, and who also had a higher threshold for referral obligations because of the lack of local specialists. However, in the modern era of communications via the Internet with e-Journals, video continuing education (CE) programs, and other technological advances, the law today recognizes a national standard of care and the local standard of care seldom provides a defense, except for possible referral threshold issues in some rare cases. Therefore the prudent practitioner must remain current on the standards of care being described in readily available CE courses, studies, journals, and texts.

## UNPROFESSIONAL CONDUCT

One area of exception to the traditional expert witness-defined legal standards of care is regulations set forth by any particular state's licensing agency, violation of which is

typically referred to as *unprofessional conduct*. Such regulations typically include the scope of practice (i.e., what any health care provider is allowed to do as determined by licensure). These regulations, while generally similar, can be very specific to individual states. For example, in California only dentists may prescribe and fit dentures. However, in Oregon, that work may be done by denturists. Typically, regulations, such as abuse reporting and Good Samaritan defenses, will vary from state to state. Other examples include record keeping regulations, which may be nearly silent in some states to very specific. Minnesota requires the use of designated forms. Violation of such regulations is considered unprofessional conduct and a form of negligence and expert testimony may not be required to prove such violations:

<http://www.comda.ca.gov/conduct.html>.

## VIOLATIONS OF STATUTES

The other exception to the traditional expert witness-defined legal standards of care is a violation of statute that may not be included in licensing regulations. Those statutes typically are found in penal and/or civil codes, and in some cases may be in Federal codes. Violation of a statute intended to regulate the practice of health care or dentistry is presumed to be evidence of negligence, and expert testimony is not required. Rather, the defendant health care provider has the obligation of trying to prove the *absence* of negligence. Examples would be statutes regarding the record keeping for medications. If a patient had a problem with a treatment involving the dosing of a medication and the defendant's record keeping was not in compliance with a statute, the treatment is presumed negligent if the harm was what the statute was intended to prevent:

<http://www.courts.michigan.gov/mcji/negligenceCh10-19/negligence-ch12.htm>.

**TIP:** It is important for the prudent practitioner to maintain basic knowledge of the laws and regulations regarding treatment and record keeping for all patients, including trauma patients. Overviews of those laws can often be obtained from dental societies, malpractice insurance carriers, and state licensing agencies.

## CONSENT ISSUES

Before providing any treatment to a patient, a licensed health care provider must obtain the consent of the patient except in cases of dental emergencies in which any delay in treatment would result in irreversible harm or injury. Failure to obtain consent before providing treatment is considered battery (unlawful touching), not a form of negligence or substandard care. Some courts have rules that claims of treatment without consent are not subject to the limits of health care tort reform, such as limits on verdicts or attorneys' fees.

In most cases, consent for an examination or treatment is implied from the mere fact that a patient presented themselves for examination or treatment, as long as they are competent to give such consent. Competency is determined under the law by either patient age or mental capacity.

Minors (persons under the age of 18 years) are generally presumed to be incompetent to give consent to dental treatment. Unless an exception applies, consent for minors can only be given by a parent (custodial parent in cases of divorce) or legally appointed guardian (relative, foster parent, or social worker). Obtaining the consent of a legal guardian is often difficult in trauma cases in which the patient is seen on an urgent basis and accompanied by an older sibling or nonguardian relative. Reasonable efforts must be made (and well documented) to contact the appropriate person for consent. In trauma cases, evaluation of the urgency of treatment is critical when contact with persons authorized to give consent cannot be reasonably obtained. In such cases, if palliative care (antibiotics and pain medications) would address the patient's pain, and if the indicated treatment could be delayed for a few days until the consent of a parent or legal guardian could be obtained, then treatment without appropriate consent would be considered a battery. However, if it is determined, and well documented, that even a short delay of care would result in irreversible harm to the minor patient's dental and/or medical health, and if palliative care would not be appropriate, then treatment may be performed without ideal consent.

Consent is also a potential problem in cases of a patient who may be incompetent as a result of a decreased mental capacity, such as disease (e.g., Alzheimer's), developmental disability (e.g., Down syndrome), injury (e.g., traumatic brain damage or chronic substance abuse), or transient conditions (e.g., drug or alcohol intoxication). In such cases, consent and treatment issues should be evaluated in the same manner as for minor patients. Consent for treatment needs to be obtained from the legal guardian. If such consent cannot be obtained, the urgency of the treatment must be carefully evaluated and documented before proceeding with care with less than ideal consent:

[http://www.intrepidresources.com/html/informed\\_consent.html](http://www.intrepidresources.com/html/informed_consent.html).

## INFORMED CONSENT/REFUSAL

There are two exceptions to the standards of care that do not require either expert testimony or violation of a statute or regulation: informed consent and the new doctrine of informed refusal.

### Informed Consent

In all states, a dentist is required to obtain an informed consent before providing dental treatment. Generally stated, a doctor has the obligation to advise the patient of the risks of serious injury or death, the potential benefits, and the reasonable alternatives to any proposed dental treatment or

therapy. The only exceptions would be in either Good Samaritan situations (see p. 186), or in cases of unconscious adult patients who would suffer irreversible and significant harm if not provided emergent therapy or treatment.

The extent and detail required of an informed consent documentation are determined by making a comparison of the necessity of the treatment, the reasonable alternatives, and the potential risks of such treatment(s). In cases of elective treatments in which reasonable alternatives include no treatment at all, the risks and complications must be discussed and documented in much more detail than treatments that are considered necessary and have few or no reasonable alternatives, and the risks are rare or remote.

### Informed Refusal

A corollary doctrine is that of informed refusal, which requires advising a patient of the risks of not having treatment, or not taking a medication, or not following a referral. That doctrine has evolved to include the obligation to advise patients of the ideal treatment(s), regardless of insurance coverage, and the risks of selecting a less than ideal treatment simply because it may be covered by insurance. Indeed in California, the state's Judicial Council (Fig. 9-1) promulgated specific instructions that have been designed for juries in cases involving claims of a lack of informed refusal. The instructions begin with a statement of the law (#534) (Box 9-1). In the event a California jury is asked to determine if there was a lack of informed refusal, they are given instructions regarding the essential elements they must consider in trying to reach a verdict (#535) (Box 9-2). For example, consider a case in which a trauma patient is seen for a fractured tooth, whereby the ideal treatment would be root canal treatment, crown lengthening, and a full coverage crown. However, the patient's insurance (private or public) would only cover an extraction followed by either a remov-

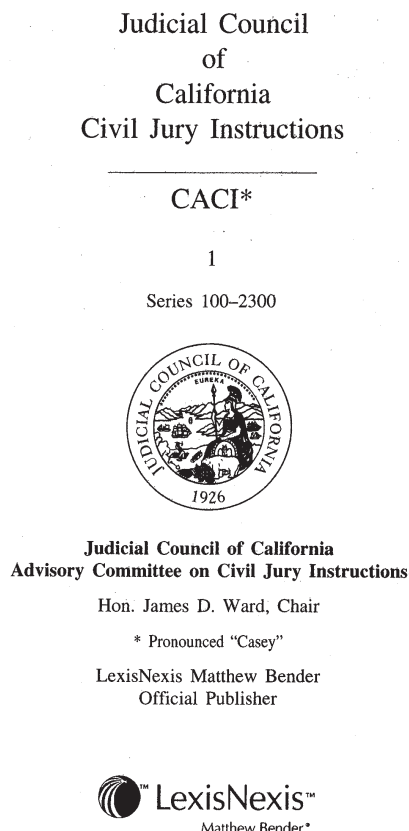
#### BOX 9-1

#### Informed Refusal— Definitions (CACI No. 534)

A [insert type of medical practitioner] must explain the risks of refusing a procedure in language that the patient can understand and give the patient as much information as [he/she] needs to make an informed decision, including any risk that a reasonable person would consider important in deciding not to have a [insert medical procedure]. The patient must be told about any risk of death or serious injury or significant potential complications that may occur if the procedure is refused. A [insert type of medical practitioner] is not required to explain minor risks that are not likely to occur.

From the Judicial Council of California Civil Jury Instructions (2006), © 2005 by the Judicial Council of California. Published 2005 Matthew Bender & Co Inc, a member of the LexisNexis Group.





This version provided by LexisNexis® Matthew Bender®, Official Publisher, 800-533-1637, [www.lexisnexis.com/bookstore](http://www.lexisnexis.com/bookstore), for public and internal court use.

**Figure 9-1** Judicial Council of California Civil Jury Instructions. © 2005 by the Judicial Council of California.

able or fixed prosthesis. It would be considered substandard care, or negligence, to fail to advise the patient of the risks (such as bone loss, injury from preparation of abutment teeth, or difficult periodontal maintenance) of selecting the lesser but insurance-covered treatment in comparison with the more ideal treatment of retaining the single tooth—regardless of lack of insurance coverage. Suggesting that the patient would not have chosen any treatment not paid for by insurance is not a defense against a claim of failure to provide informed refusal.

**TIP:** Some form of documentation of the informed consent/refusal discussion should be made before any treatment is performed. Such documentation may be as simple as a chart entry that the risks, benefits, and alternatives (RBAs) of dental treatment were discussed or as detailed as a patient-signed form discussing the risks of apical surgery. Studies have shown that patients often forget the informed consent discussion and believe it did not take place. Therefore documentation by way of signed consent forms, while generally not required by the law, is highly recommended by risk managers and insurance carriers. Some dental specialty

## BOX 9-2

### Risks of Nontreatment—Essential Factual Elements (CACI No. 535)

[Name of plaintiff] claims that [name of defendant] was negligent because [he/she] did not fully inform [name of plaintiff] about the risks of refusing the [insert medical procedure]. To establish this claim, [name of plaintiff] must prove all of the following:

1. That [name of defendant] did not perform the [insert medical procedure] on [name of plaintiff];
2. That [name of defendant] did not fully inform [name of plaintiff] about the risks of refusing the [insert medical procedure];
3. That a reasonable person in [name of plaintiff]'s position would have agreed to the [insert medical procedure] if he or she had been fully informed about these risks; and
4. That [name of plaintiff] was harmed by the failure to have the [insert medical procedure] performed.

From the Judicial Council of California Civil Jury Instructions (2006), © 2005 by the Judicial Council of California. Published 2005, Matthew Bender & Co Inc, a member of the LexisNexis Group.

associations and some malpractice insurance carriers provide sample consent forms, generally based upon areas where claims are more frequent:

<http://www.redwoodsgroup.com/default2.asp>.

## BURDEN OF PROOF

The *burden of proof* in a civil malpractice case is measured as a *preponderance*, or a simple majority (more than 50%), of the evidence, not *beyond a reasonable doubt* (more than 90% of the evidence) as in criminal cases. Therefore the detail and quality of dental records are essential in reducing the risks of being subject to a malpractice claim.

## CAUSATION

In order for a patient to win a malpractice suit, they must prove *causation*, which is evidence of substandard care that probably caused some injury. As with the standard of care, most of the evidence comes by way of expert testimony.

## CONDUCT OF OTHERS

Under the law, for a patient to recover damages, the malpractice need not be the sole cause of the injury, but merely a substantial factor if there are other causes. Those other

causes can even be the negligence of other persons or entities. A typical example would be where a dentist sees a trauma patient, diagnoses injury to the pulp of a lower molar, performs root canal treatment during which a file breaks and during retrieval attempts, the broken segment is pushed out of the apex. The patient is then referred to an oral surgeon for apical surgery. During that surgery, the surgeon drills into the mandibular canal and severs the inferior alveolar nerve. In such a case, the patient need only bring a claim or suit against the first dentist to recover damages for the nerve damage.

Two principles are applicable in this example. First, basic tort law holds that a negligent person is liable for all the consequences of that negligence, including exposure to the negligence of others. Therefore in such cases, the patient need only bring suit against the first health care provider. Second, the law in most states provides for apportionment of fault between negligent persons, even ones that are not named defendants in a suit. Generally the amount of the apportionment that may be recovered against any one defendant may be determined by the nature of the damages to be apportioned. In many states, general damages (pain and suffering) are apportioned and awarded by a percent of fault, whereas 100% of special damages (expenses such as medical bills and wage loss) may be recovered from any defendant, and that defendant may then seek indemnity against the other defendants. However, those indemnity rights might be meaningless if the other defendants have no insurance or no assets. Such situations are commonly referred to as “the deep pocket” rule.

**TIP:** Try to only refer patients to health care providers with whom you have a good relationship and have good knowledge of the quality of their practice, including record keeping. Ideally, those practitioners should have malpractice insurance coverage to avoid becoming victimized by the deep pocket rule.

## GOOD FAITH SETTLEMENT

The law encourages resolution short of trial, and therefore most states have incentives known as *good faith settlements*. A settlement is the payment of money by a defendant to the plaintiff in exchange for dropping the lawsuit. There is no implied or assumed admission of fault as a result of a settlement or offer of settlement.

In the event a defendant settles a lawsuit short of trial, and a court finds and orders the amount to be in *good faith*, then the other defendants are barred from seeking indemnity from the settling defendant, regardless of the type or size of any subsequent verdict against the nonsettling defendants.

Another incentive involves resolutions before formal litigation. In the event a health care provider offers a settlement to a patient complaining of a bad result or threatening a lawsuit, and the offer is rejected, that offer cannot be later used or even mentioned in a lawsuit by that same patient.

However, if the offer is accepted, a lawsuit is avoided. The only exception to this rule would be if the offer of settlement were accompanied by an explicit admission of fault or wrong doing.

**TIP:** Time lost from work by a dentist defending a lawsuit can result in the loss of thousands, and in some cases, tens of thousands of dollars. Recognizing that health care is as much a business as a profession, offering a patient a negotiated settlement is sound business, a guaranteed result, and much less stressful than fighting a lawsuit and being cross-examined by an attorney. Consider such an offer with patients who had sustained a very bad result or where it took much more time and expense to accomplish a reasonable result through no fault of the patient. When offering a refund or settlement, do not acknowledge fault or wrong doing. Rather, advise the patient that their concerns and complaints are recognized and the offer of payment is merely a way of addressing those concerns and trying to stand behind one's work.

## NATURE OF INJURY

In the event of a finding of substandard care, damages can be awarded for any physical, mental/emotional, and/or financial injuries sustained by a patient-plaintiff. The fact that a patient may have a rare or unusual reaction to substandard care is no defense. The law is that you “take the victim as you find him.”

**TIP:** Do not make exceptions in your patient information gathering methodology (dental and health history) just because of a nonscheduled appointment for a trauma patient. Indeed the prudent practitioner will require completed, dated, and signed written medical and dental history before performing any treatment, even to an emergency patient. Also because of the dynamic nature of diseases and their treatments, history forms should be updated every 2 years to be sure the questions remain relevant and current.

## NATURE OF THE TRAUMA

In the case of treatment of a trauma patient, the standard of care and associated legal requirements will vary depending upon the nature of the trauma. Traumatized patients generally fall into three categories: accident/sports, abuse/neglect, or iatrogenic procedures (dental or medical).

In addition to quality records and radiographs, digital imaging is one of the best ways to document trauma. An intraoral digital wand style camera is ideal. The next best system would be a “through the lens (view/focus)” camera with a macro lens, followed by a reflex camera with a macro setting. Photography is particularly important in situations in which the evidence of trauma might be altered or may fade, such as a cracked tooth that might be restored or bruised.

ing (evidence of abuse) that might fade. Also, photography can be an effective way to record evidence that can be attached to insurance submissions to validate the necessity of treatment.

**TIP:** Judicial acceptance of digital records (notes, radiographs, and photographs) often depends upon the way the evidence is maintained. In the case of digital records, be sure to use a system that prohibits alterations, such as additions or deletions after the close of business each day. When taking radiographs or photographs, the chart (paper or digital) should have notations as to the number of images and the respective views. Any image changes or enhancements should be saved as a separate file so as to not overwrite the original file, thereby changing the date of the image and creating a conflict in the records. Undocumented changes can expose the doctor to a claim of fraudulent records.

## STANDARDS FOR TRAUMA CASES

The standards for patient evaluation, treatment, and record keeping in trauma cases, as compared with regular or scheduled patients, involve urgency of evaluation, treatment, or referrals. However, basic information gathering cannot be compromised. Failure to take a full and complete medical history before devising a treatment plan, performing treatment, or prescribing a medication is below the standard of care with one basic exception. In those cases in which even a short delay in treatment would result in significant dental harm, and the risks of inadequate dental/health history are low, the law allows for treatment. For example, when a patient has an avulsed tooth, immediate reimplantation may be appropriate in cases in which the time or situation does not allow for a full dental/medical history. In such cases, a full health history should be obtained as soon as reasonably possible, particularly before any medications, such as antibiotics or analgesics, are prescribed. Referrals and reports to other practitioners should be carefully documented. In the event photographs and/or radiographs are taken, they should be shared with other practitioners who will be continuing or taking over the care of the patient. Failure to transmit such information increases the potential for errors in treatment, such as therapy on the wrong tooth, by other offices. In such cases in which a suit is filed by a patient, it is common to include the referring doctor as a defendant in addition to the doctor who performed the treatment.

## GOOD SAMARITAN DEFENSES

In years past, health care providers often hesitated to engage in providing emergency services to trauma patients at other health care offices (such as being called to another office in the same complex) or outside any health care setting because of concerns about potential liability. As a result, all states

have passed Good Samaritan laws. Indeed even Canada passed such laws (Civil Code of Québec, Book Five, art. 1471), which state that anyone who, in good faith, helps a person in danger is protected from legal proceedings. An individual may not be held responsible for injury caused while assisting a person in danger unless the injury is caused by his intentional or gross fault. Those laws provide that in cases in which a health care provider is called in to provide care on an urgent basis to a trauma patient outside of normal office settings, such a practitioner cannot be held liable for violations of the standard of care except in cases of gross negligence or intentional conduct.

However, the Good Samaritan rule does not apply to cases involving treatment of trauma patients in the doctor's own office. In those cases, the standard of care is the same as with the nontrauma patients. Another exception includes dentists who routinely offer and perform their services at other offices, such as a visiting endodontist or dental anesthesiologist.

**TIP:** When providing urgent care outside of your own dental office, be sure to create a separate record of the events indicating:

- How you were called to provide urgent services outside of your offices
- The patient condition observed
- Basic diagnosis considered
- Treatment provided

## NONINTENTIONAL INJURY/ACCIDENT/SPORTING CASES

Nonintentional injury or accident and sporting trauma cases typically present some urgency in diagnosis and treatment that may minimize the opportunity for a complete work-up. The degree to which patient work-up protocols may be compromised and still comply with the standard of care will depend upon a balancing of the harm that may occur in the absence of immediate care as compared with the harm that might occur because of a less than complete work-up.

In cases of avulsed teeth, time is typically of the essence, and reimplantation is generally considered urgent. Documentation of the events and time frame of the avulsion will provide justification for a less than ideal work-up.

In addition to treatment of the obvious effects of trauma, the standard of care requires documentation of an evaluation for potential pathological conditions that may not have distinct or clinical symptoms, such as bone fractures or temporomandibular joint (TMJ) injury. Therefore careful documentation of the nature of the trauma, including the source and areas impacted, is essential. Subsequent imaging should be considered and documented to rule out subclinical injury. Also, depending upon the nature of the trauma reported, a TMJ examination should be considered and if performed, so documented:



[http://www.redwoodsgroup.com/dentistry/rmt\\_commonemergencies.asp](http://www.redwoodsgroup.com/dentistry/rmt_commonemergencies.asp).

**TIP:** Many malpractice suits involving dental trauma will allege missed diagnosis of pathological conditions, such as fractures or TMJ injury, because of a tendency of dentists to only chart positive findings. The claim in such cases is that the doctor did not perform a complete examination. To minimize the potential for such suits, routinely chart the absence of problems, such as:

TMJ—WNL, 2-PAs No Fx.

When performing pulp testing, record the negative and the positive findings.

## ABUSE AND BATTERY CASES

In all dental examinations, and particularly in trauma cases, the laws of all states require that dentists be observant of the potential for abuse, physical and/or mental, of children, the disabled, or the elderly. Upon obtaining knowledge of or observing evidence of abuse, a report must be made to the appropriate governmental agency. The mandating federal law is known as the *Child Abuse Prevention and Treatment Act* (CAPTA) (Jan. 1996 version). In the case of minors, reports should be made to Child Protective Services. In the case of adults of any age, reports are made to the local police department, some of which have abuse investigation units:

[http://nccanch.acf.hhs.gov/pubs/reslist/rl\\_dsp.cfm?rs\\_id=5&rate\\_chno=11-11172](http://nccanch.acf.hhs.gov/pubs/reslist/rl_dsp.cfm?rs_id=5&rate_chno=11-11172).

## CONFIDENTIALITY AND PRIVILEGES

Most state statutes provide that patient confidentiality laws are overridden by abuse reporting laws. The HIPAA Privacy Rule permits doctors to disclose reports of child abuse or neglect to the appropriate government authority:

[http://www.redwoodsgroup.com/dentistry/dental\\_HIPAA.asp](http://www.redwoodsgroup.com/dentistry/dental_HIPAA.asp).

## IMMUNITY

CAPTA requires states to enact legislation that provides for immunity from prosecution arising out of the good faith reporting of abuse or neglect. In most states, a person who reports suspected child abuse in “good faith” is absolutely immune from criminal and civil liability. Statutes describe good faith as a “reasonable cause to believe” or a “reasonable suspicion.” Other statutes require the doctor to “know or suspect.” However, under any standard, immunity is limited. CAPTA requires states to enact legislation providing for prosecution in false reporting cases (reports without having a reasonable belief that the report is true).

**TIP:** Document clinical evidence of abuse with descriptive charting, victim statements, and/or photographs where applicable. Quality documentation will support a good faith reporting of abuse in any state. More importantly, failure to report can have much more significant consequences.

## FAILURE TO REPORT

Failure to report suspected child abuse can result in criminal prosecution, generally a misdemeanor punishable by a fine. However in severe cases, such as when a dentist does not report suspected abuse and the victim is killed by the abuser, some prosecutors have asked for jail sentences for the dentist. In addition, state licensing agencies often consider failure to report abuse as unprofessional conduct, subjecting the dentist to licensure sanctions. More importantly, failure to report can result in civil liability, a lawsuit asking for monetary damages. Such claims can be brought by either the victim or relatives of the victim. For example, in the case of a child or an elderly person, if the dentist were to fail to report clinically apparent evidence suggestive of abuse, and if the child or elderly person were subsequently to be killed by their abuser, a claim for wrongful death could be brought against the nonreporting dentist, just as if that dentist caused the death during negligent dental care. Also because failing to report abuse is a violation of a statute, it is presumed that the injury (death) was caused by negligence of the dentist, and no expert testimony is required.

## RECOGNITION OF ABUSE

### Child Abuse

*Child abuse* generally falls into three categories: severe neglect, and mental and physical abuse:

<http://nccanch.acf.hhs.gov/pubs/factsheets/whatiscan.cfm>.

Evidence of physical trauma or extensive neglect should be carefully documented, including the results of questioning of both the child and parent or guardian regarding the source or reasons for the conditions observed.

Complicating issues are religious beliefs that may result in resistance to important or necessary care. However, the constitutional guarantee of freedom of religion does not allow for parental neglect or intentional withholding of necessary dental care:

<http://www.cirp.org/library/ethics/AAP2/>.

### Abuse of the Elderly or Disabled

*Abuse of the elderly or disabled* is less commonly seen in a dental office due to assisted living and nursing home care for the elderly:

<http://www.asaaps.org/basics/index.php>.

Not all states mandate health care provider reporting of elder abuse:

<http://www.abanet.org/media/factbooks/eldt2.html>.

However, those that do most often make that requirement when it becomes clinically apparent that the patient is dysfunctional and unable to report the abuse themselves; few states specifically include dentists in their list of professionals who are required to make reports:

[http://66.102.7.104/search?q=cache:AVMgaqHY\\_PIJ:www.ndaaapri.org/apri/programs/senior\\_fraud/EA\\_MSR\\_](http://66.102.7.104/search?q=cache:AVMgaqHY_PIJ:www.ndaaapri.org/apri/programs/senior_fraud/EA_MSR_)

[long\\_final\\_5\\_29\\_2003.doc+disabled+abuse+mandatory+reporting&hl=en](#).

**TIP:** Because of the potential exposure for failure to report abuse and because most of the mandatory reporting statutes provide immunity for good faith reporting, the prudent dentist should err on the side of reporting cases of suspected abuse to the appropriate agency. Indeed, most such agencies have hot lines that will provide some guidance as to whether or not the observed conditions need to be reported:

<http://www.asaaps.org/basics/reporting.php> (adults)  
<http://www.focusas.com/Abuse.html> (children).

## IATROGENIC TRAUMA CASES

Iatrogenic injury (induced in a patient by a dentist's activity, manner, or therapy) presents some legal and ethical issues.

In cases in which iatrogenic injury is a risk of treatment or therapy, the law of informed consent, discussed previously, applies. For example, consider a case in which removal of decayed or damaged tooth structure creates a pulp exposure. In such cases, exposure of the pulp is often very treatable:

[http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list\\_uids=2032743&dopt=Abstract](http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?cmd=Retrieve&db=PubMed&list_uids=2032743&dopt=Abstract).

However, typically endodontic treatment requires a full coverage restoration, and the patient may not have the means or desire to have or fund such treatment. Therefore failing to obtain informed consent before performing the exposure-prone treatment would be below the standard of care. Any patient may choose to have a tooth extracted rather than risk pulp exposure.

**TIP:** When clinical and radiographic evidence suggests a potential for pulp exposure during removal or repair of decayed or damaged tooth structure, patients should be advised of the risk of pulp exposure, the consequential endodontic and restorative therapy, and the costs of both, and then advised of the reasonable alternatives, including extraction without replacement. Documentation may be as simple as:

*"P—advised of Fx cusp, Rx Inlay, RB&A discussed (a common abbreviation for Risks, Benefits, and Alternatives)—P OK'd."*

Signed informed consent is not typically indicated for routine restorative treatment. If a patient declines a recommended treatment, the record should reflect obtaining *informed refusal*. Documentation may be as simple as:

*"P—advised of need for RCT #19, P declines—RB&A discussed, P—still declines—insists on ext. Referral to OMS."*

To fortify the note, the patient can be asked to sign the chart entry, but in such cases the abbreviations should be expanded into full text and lay terms.

*Errors* in treatment present legal and ethical issues. The law does not require a dentist to advise a patient of substandard treatment unless there is a certainty of the manner and environment of the prior treatment (e.g., redoing previously done dental treatment that was of poor quality, whereby the original inappropriate treatment potentially compromises the desired future treatment). This author has been defending dentists for more than 30 years. Typically experts are hired to determine if there are breaches in the standard of care. Despite extensive review of numerous records, analysis of hours of deposition testimony, and imaging evidence, it is often difficult for expert witnesses to fully reconstruct the facts and environment surrounding treatment and complications to determine if there was a breach of the standard of care. Therefore it is presumptuous for a treating dentist to opine on standard of care issues when only having the patient to examine, the patient's version of the past treatment, and few or no records. Indeed the ADA Code of Ethics 4-C-1 defines "justifiable criticisms" to exclude differences in opinions as to the manner of treating a particular condition:

[http://www.ada.org/prof/prac/law/code/principles\\_04.asp#4c](http://www.ada.org/prof/prac/law/code/principles_04.asp#4c).

*Equipment problems* are a growing area of concern as the technology of dentistry grows at an increasing rate. To minimize iatrogenic injury from equipment malfunction, dentists should know and follow routine maintenance schedules, documenting those efforts.

*Staff management* includes documented training of staff in both the use and maintenance of dental technology. In the event of iatrogenic injury resulting from equipment failure or breakdown, the injured patient may introduce evidence of the equipment manufacturer's maintenance guidelines and any failure of staff or the dentist to document adherence to such schedules.

Many states (like California) require *reporting* a death or immediate hospitalization of a patient as a result of dental treatment. In such cases, the licensing board may investigate the event, including a review of records and interview of the dentist and staff. Failure to make such a report is substandard care (California Business and Professions Code section 1680-Z).

Also, in this author's experience, most catastrophic claims of substandard care result not so much from the treatment that caused the problem, but rather from the lack of timely and efficient response to the complication.

**TIP:** In the event of any catastrophic event associated with dental care, contact your insurance carrier's hot line for advice and information on reporting statutes. Often, early intervention can minimize the potential for communication failures inside and outside of the office.

Regarding dental emergencies, have a well-established set of protocols for staff and doctor's response, and rehearse those protocols at least once or twice a year.

## CONCLUSION

Dental trauma cases present some unique and routine legal and risk management issues for dentists. Rigorous attention to detailed and accurate records remains the best method for avoiding claims of substandard care. Quality evidence gathering and maintenance are essential and should include use of both radiographic and photographic imaging. Maintaining records and graphic evidence require staff whose training is routinely evaluated and updated for quality, accuracy, and compliance with office policy.

Record keeping should be uniform for staff and dentists. Digital dentistry and records will increase the detail and

coverage of records, while reducing the time required to record the data.

The differential diagnostic process should be well documented, unless there is a significant urgency of treatment, and then the nature and environment of the trauma must be well documented. Once treatment plans are designed and discussed with the patient, parent, or guardian, informed consent and/or refusal should be documented.

Staying current with the legal requirements for trauma patients will allow today's dental practitioner to enjoy the rewards of helping such patients and still avoid the risks of a claim of substandard care.



# THE PSYCHOLOGICAL IMPACT OF DENTAL INJURIES

■■■  
HAL LIPTON

## CHAPTER OUTLINE

### THE LITERATURE ON PSYCHOLOGICAL REACTIONS TO PHYSICAL INJURIES

#### REVIEWING EMOTIONAL AND BEHAVIORAL SYMPTOMS THAT MAY ACCOMPANY DENTAL INJURIES

Acute Stress Disorder

Posttraumatic Stress Disorder

Other Signs of Distress

#### IMPLICATIONS FOR THE DENTIST IN PROVIDING SUPPORT TO A PSYCHOLOGICALLY AFFECTED PATIENT

#### CONCLUSION

In recent decades, trauma professionals and mental health clinicians have become keenly aware of the potential for adverse psychological reactions following auto crashes, multiple trauma, combat injuries, rape, natural disasters, physical assaults, and other shocking events. Trauma team members have learned that a full recovery from serious injuries requires attention to both the physical and psychological needs of the patient. But can a patient with only a “simple” dental injury suffer from intense and disturbing emotional reactions following his or her injury? What are the potential emotional reactions to sudden and shocking events? How can the dentist recognize an injured patient’s adverse emotional reactions to a dental injury? How can the dentist be helpful to adults and children emotionally traumatized by injuries to the mouth and teeth? This chapter will offer perspectives on the questions raised.

## THE LITERATURE ON PSYCHOLOGICAL REACTIONS TO PHYSICAL INJURIES

Investigators<sup>7</sup> write that there have been few studies that examined specific posttraumatic psychological effects of facial injury. They point out that unless recognized and treated, posttraumatic psychological problems can become chronic. Clinicians should be aware of presenting features (symptoms) and that treatment options may include referral to mental health services. For patients averse to using mental health specialists, the authors indicate there is evidence for the use of antidepressants in dealing with serious psychological reactions.

Another study<sup>8</sup> indicates that “a significant minority of those physically injured in a traumatic event will develop psychiatric disorders, including anxiety, depression, and posttraumatic stress disorder (PTSD).” They indicate that

rates of PTSD after serious accidents, including motor vehicle crashes, have ranged from 11.6% to 23.6%. Their two main conclusions are that "... physical injuries need not be great to precipitate adverse psychological sequelae, and that even individuals who have stable backgrounds and who have high levels of life contentment before their trauma can develop acute psychological distress."

Another study<sup>11</sup> points out that the psychosocial sequelae of craniofacial disfigurement may have as great an impact on the patient as the strictly physical aspects of the problem. They also suggest that very little systematic work has been focused on these effects.

Other investigators<sup>10</sup> point out that intentional violence-related injuries are apt to trigger high rates of emotional distress in their victims. They also write that "... orofacial injury is a significant, yet mostly underappreciated, aspect of the injury burden borne by our vulnerable (minority) populations." The authors state that treatment is geared entirely toward tending to the overt physical manifestations of the injury, and that psychological problems are rarely considered. They indicate that common reactions to traumatic injury include symptoms of repeated and unwanted experiencing (reexperiencing) of the event, hyperarousal, anxiety, and a persistent sense of current threat.

Investigators<sup>4</sup> studied psychological distress among trauma survivors in a large urban hospital. They found that:

*"Absolute levels of PTSD symptoms were high at 1 month (following injury); 25% of the sample appeared to meet diagnostic criteria for acute PTSD based on a self-report of symptoms. Variables associated with ... higher rates of PTSD symptoms included older age, being female, prior psychological disturbance, ... exposure to and distress at a prior trauma as well as overall high rates of stressful life events in the patient year ... and unmet social support needs during the recovery phase."*

The authors suggest that the use of screening mechanisms for PTSD may be in order for survivors of orofacial injury and that referral to appropriate psychological treatment is important for those seriously stressed.

In another investigation,<sup>5</sup> trauma surgeons studied adolescents ages 12 to 19 involved in major traumatic events. They found that the rate of long-term PTSD was 27%, with high rates over the follow-up period. Risk factors for long-term PTSD were perceived threat to life, death of a family member at the scene, no control over injury event, and violence-related injury. This suggests that dentists should be aware of the circumstances of the event, and the nature of the injury, in assessing possible adverse emotional effects of the traumatic event. Perhaps a relatively minor dental injury can become a major psychological problem for those involved in injuries suffered in particularly distressing events.

Another investigation<sup>12</sup> involved a study of traffic-injured children, aged 15 to 17 years of age, and admitted to the

hospital for treatment of injuries from traffic crashes; this study included the parents too. They found that symptoms of acute stress disorder (ASD) were commonly observed in the children and parents. Eighty-eight percent of children and 83% of parents reported having at least 1 clinically significant symptom; this affected 90% of the families. Broad distress was observed for a large minority: 28% of children and 23% of parents. The authors indicate that clinicians should be alert to identify those with persistent and distressing responses to a traumatic event experienced by an individual, children, and family, such as a traffic crash. All families require emotional support when a child is exposed to a traumatic event. Pediatric care providers can expect to see at least a few significant acute stress symptoms in most children and parents in the early aftermath of a traffic-related injury. In this situation, brief education is appropriate to explain that these are typical reactions and are likely to resolve as the physical injuries heal and as the family uses its normal coping methods to deal with the immediate shock. Pediatric providers can prepare parents to be observant of their own and their child's reactions by briefly reviewing symptoms of reexperiencing, avoidance, hyperarousal, and dissociation. Parents and children with persisting distress also may be reassured by a personal referral to a trusted behavioral health provider who specializes in treating families who have experienced a trauma.

In a study of Brazilian children, investigators<sup>2</sup> compared children with untreated or inadequately treated tooth fractures with children who had no fractured teeth. They found that children with untreated or inadequately treated permanent teeth were far less well adjusted to living than those children who had no such injuries.

Another study<sup>9</sup> suggests that the role of violence in cases of dental injuries to children may be underestimated. This study found that children subjected to bullying or being picked on were more likely to suffer from dental injuries than the children who carried out the bullying behaviors. They indicate the need for more research about the role of bullying as a significant cause of traumatic dental injury.

One investigator<sup>1</sup> suggests that dental injuries may become particularly significant in a culture so concerned about physical appearance. She points out that Hollywood continues to be a major source of obsession with appearance. For example, with the advent of television programs such as *Extreme Makeover* and *The Swan*, the public is now aware of what it takes to make them beautiful and youthful looking and may seek the advice of their dentist.

A different study<sup>6</sup> echoes the opinions of other writers about emotional responses to traumatic injuries: that the severity of traumatic injury did not predict higher rates of symptoms. These investigators suggest that if acute PTSD symptoms presage the development of chronic PTSD, early identification and treatment referral of high-risk cases are imperative. They point out that clinicians monitoring the ongoing recovery of orofacial injury can play a critical role

in this effort. These authors indicate, as do other clinicians, that acute stress symptoms usually abate or disappear within 1 month or less. Common early normal reactions to trauma include: numbness and denial, fear, depression, elation at having survived, anger, guilt, impaired sleep, perceptual changes, and flashbacks. Patients and family members need to be assured that such reactions are normal and do not usually last for more than a few days or weeks. However, if patients demonstrate an inability to accept the injury, are not compliant with treatment, experience negative changes in their social interactions or work practice, or experience excessively prolonged emotional reactions, a referral for a psychological or psychiatric assessment by a specialist may be warranted.

## REVIEWING EMOTIONAL AND BEHAVIORAL SYMPTOMS THAT MAY ACCOMPANY DENTAL INJURIES

It may be useful here to review some of the terminology pertaining to psychological reactions to physical injuries or other traumatic events, including dental injuries.

### ACUTE STRESS DISORDER

Acute stress disorder (ASD) consists of a cluster of symptoms that may include reexperiencing the event, avoidance, hyperarousal, and/or dissociation (e.g., feelings of emotional numbing or feelings of unreality).

### POSTTRAUMATIC STRESS DISORDER

Symptoms of posttraumatic stress disorder (PTSD) may include one or more of the following according to a leading trauma expert:<sup>3</sup>

#### *Physical Symptoms*

- Hypervigilance
- Exaggerated startle response
- Difficulty sleeping
- Difficulty with concentration or memory
- Mood irritability, especially anger and depression

#### *Intrusive Symptoms*

- Recurring, distressing recollections (thoughts, memories, dreams, nightmares, and flashbacks)
- Physical or psychological distress at an event that symbolizes the trauma
- Grief or survivor guilt

#### *Avoidant Symptoms*

- Avoiding specific thoughts, feelings, activities, or situations
- Diminished interest in significant activities
- Restricted range of emotions (numbness)

## OTHER SIGNS OF DISTRESS

Other signs of distress include shock, anger, depression, fear of the event happening again, guilt, lack of control, difficulties in cognitive functioning, and sleeping and eating disturbances. Adults and children can be very concerned about how they look; will their appearance be normal again? Even temporary changes in the teeth and facial areas can cause anxiety and a diminution of self-esteem. The anticipation of pain associated with dental treatments is not to be underestimated following dental injuries. Many patients are apprehensive about going to the dentist in “normal times.” But when dental injuries occur—usually shocking events—they may arouse more than normal feelings of apprehension about the dental visits. Not all dental injuries can be repaired quickly or with excellent results. For example, patients with temporomandibular disorder (TMD) may experience many problems over the years—problems that take a good deal of energy to deal with. Over the long term, teeth repaired with crowns or bridges eventually may require more dentistry—some of it being very expensive. One can expect at least some sense of disappointment and anger from those occurrences. Some specific reactions to chronic injuries include anger and depression.

Dentists should be aware of the short-term but troubling consequences, should the patient’s injuries mar their appearance. School presentations, job interviews, oral reports, public speaking, social situations, and other events may cause embarrassment, humiliation, avoidance, and anger. In the overall scheme of life, these reactions may seem minor. But to the troubled patient, they may be major concerns of the moment.

## IMPLICATIONS FOR THE DENTIST IN PROVIDING SUPPORT TO A PSYCHOLOGICALLY AFFECTED PATIENT

The list of possible psychological problems resulting from physical injuries, including dental trauma, is long—perhaps overwhelming. Considering what to do about such possible reactions to the emotionally traumatized can be daunting. After all, the busy dentist is not a psychologist nor does he/she have a great deal of time to deal with the emotional trauma of patients.

The good news is that the majority of dentally injured patients will be able to handle any reactions from their injuries quite well. They usually share their negative reactions with family or friends before visiting the dentist. Those that suffered multiple injuries or other horrific events usually have support from trauma and mental health clinicians in trauma centers. However, it should not be surprising that dentally injured patients, both adults and children, can benefit from additional support from their dentist. Such



support may only require a few moments of inquiry, identification of symptoms, provision of information, and reassurance that the injuries can be fixed and any discomfort experienced will be minimized. The duration of providing emotional support to the patient need not be long—it is the quality of caring and concern provided that can have the desired impact on the patient's ability to cope with his or her emotional distress. In a few moments, the dentist usually uncovers severe emotional reactions to injuries. The identification of distress can be made by observation of the patient's affect and behavior, and by obtaining a brief history of the event that caused the physical injuries. For example, the dentist may gently inquire in a soothing and caring way with questions such as:

- What caused the injury? What happened?
- Did you only hurt your teeth or did you suffer other injuries?
- How badly were you hurt?
- Were you hospitalized?
- Was anybody else badly injured?

When the patient reveals responses to the questions of the dentist, it is important to show both understanding and caring:

- Oh that must have been a nightmare for you . . .
- What you went through is awful . . .
- You must have been shocked . . .
- Did you ever experience something like that before?
- What is the worst thing about your injury?
- You should know that what you have experienced is very normal. Patients who go through what you have often have dreams or nightmares and are anxious for several days or weeks. There can be other symptoms of distress. Again, all of these symptoms are normal and usually disappear in a few days or weeks . . . however, they can feel awful . . .

A few moments of dialogue with the patient is usually sufficient for the caring attitude of the dentist to come through. Information about the current injuries also has to be addressed. Dentists are familiar with informing the patient about the problems that need to be corrected and what to expect in treatment in noninjury care. In the dental injury scenario, the dentist may need to be especially concerned about how he or she will try to minimize the pain that may accompany treatment—the injuries already have caused pain—extra reassurance about pain mitigation will be appreciated. Giving clear information and answering questions also helps the patient to regain some small sense of control over his or her life. Letting the patient know that the appearance can be restored or improved is most important. Patients do not always share their worries, concerns, and apprehensions—even about appearance. But in nearly all cases it would be normal to be concerned about one's looks. When discussing the patient's emotional and dental status, it would help if the dentist would sit at eye level with the patient. This face-to-face discussion can convey a caring attitude and a feeling that the dentist is not rushing through the

emotion-laden conversation. Indeed the patient will be pleased and reassured that the dentist is really concerned and understanding. Mental health clinicians know that even their skilled interventions do not always result in a full “recovery” of the patient's coping abilities following traumatic events. Dentists need not worry about providing a complete solution to one's psychological reactions to dental trauma—active listening, caring, giving information, and answering questions go a long way toward helping the patient cope with any traumatic dental injuries. If any patient continues to be highly distressed by intensive or intrusive thoughts, the dentist may refer that patient to a psychologist, social worker, or psychiatrist who specializes in recovery from traumatic events.

If the patient is a child, support will be needed by both the patient and the parents. Children need to know that:

- Dental injuries occur sometimes during play or in accidents
- Injuries can happen to all kids
- The dental problems can be fixed
- The patient can look as good as new in a short time

It sometimes helps if the parents allow the child to bring a favorite toy or stuffed animal with them to the dental visit. Adolescents may be especially sensitive to their appearance and need to know that their looks and/or smile can be restored after injuries. The dental office staff needs to be especially sensitive to injured children and can provide reassurance and distraction as necessary.

Parents need to know that the dentist is not judging them as “bad parents” because an injury has occurred. Parents have a way of blaming themselves for the injuries of their children. They need to know that childhood injuries are common and that even the best parents in the world cannot prevent all accidents or injuries. If the parents are too hard on the child for causing the injury, or embarrassed by the child's stressful symptoms, the dentist can “normalize” childhood injuries and any stress reactions that may be present. Parents and children need to know that various stress reactions may be expected after sudden injury. A follow-up phone call from the dentist to the family, checking on their emotional progress following a traumatic event, is a much-appreciated step. If the child or parent is suffering from an intense or disturbing reaction to a dental injury—one that does not abate after a few days or weeks—a referral to a mental health professional may be indicated.

## CONCLUSION

For any patient that has incurred any type of physical trauma, the holistic management of the patient must include psychological considerations. There are cosmetic concerns of the patient and the potential loss of function. Often when children are involved, the parents are important partners in the emotional support of these traumatized patients. The parent needs to be counseled as to how to best help their

child. As the provider of dental care for patients who have undergone dental trauma, it is important that the dentist recognize the emotional state of these patients and provide the necessary support.

## REFERENCES

1. Arcan S: Overview of facial cosmetic surgery, *California Dent Assoc J* 32;10:849-853, 2004.
2. Cortes M, Wagner M, Sheiham A: Impact of traumatic injuries to the permanent teeth on the oral health-related quality of life in 12 to 14 year old children, *Community Dent Oral Epidemiol* 20(3):93, 2003.
3. Flannery R: Posttraumatic stress disorder: the victim's guide to healing and recovery, Crossroad, 2001, New York.
4. Glynn A, Asarnow J, Asarnow R et al: The development of acute posttraumatic stress disorder after orofacial injury: a prospective study in a large urban hospital, American Association of Oral and Maxillofacial Surgeons, *J Oral Maxillofac Surg* 61:785-792, 2003.
5. Holbrook T, Hoyt D, Coimbra R et al. Long-term posttraumatic stress disorder persists after major trauma in adolescents: new data on risk factors and functional outcome, *J Trauma Inj Infect Crit Care* 58:764-771, 2005.
6. Hull A, Lowe T, Finlay P: The psychological impact of maxillofacial trauma: an overview of reactions to trauma, *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 95:515-520, 2003.
7. Hull A, Lowe T, Devlin M et al: Psychological consequences of maxillofacial trauma: a preliminary study, *Br Assoc Oral Maxillofac Surg* 41:317-322, 2003.
8. Joy D, Probert R, Bisson J et al: Posttraumatic stress reactions after injury, *J Trauma Inj Infect Crit Care* 48(3): 2000.
9. Odoi R, Croucher R, Wong F et al: The relationship between problem behavior and traumatic dental injury amongst children aged 7-15 years old, *Community Dent Oral Epidemiol* 30:392-396, 2002.
10. Shetty V, Glynn S, Brown KS: Psychosocial sequelae and correlates of orofacial injury, *Dent Clin North Am* 47:141-157, 2003.
11. Stricker G, Clifford E, Cohen LK et al: Psychosocial aspects of craniofacial disfigurement. A "State of the Art" assessment conducted by the Craniofacial Anomalies Program Branch, The National Institute of Dental Research, *Am J Orthod* 76(4):410-422, 1979.
12. Winston F, Kassam-Adams N, Vivarelli-O'Neill et al: Acute stress disorder symptoms in children and their parents after pediatric traffic injury, *Pediatrics* 109(6):e90, 2002.

## OROFACIAL TRAUMA PREVENTION



RAY R. PADILLA

### CHAPTER OUTLINE

#### ATHLETIC MOUTHGUARDS

History of Mouthguards

Types of Athletic Mouthguards

Stock Mouthguards

Boil and Bite Mouthguards

Custom-Made Mouthguards

Vacuum Thermoformed Mouthguard

Pressure Laminated Mouthguard

#### ORTHODONTIC AND MIXED DENTITION CONSIDERATIONS

#### COMPLIANCE AND ACCEPTANCE OF MOUTHGUARDS

#### ROLE OF THE DENTIST IN OROFACIAL TRAUMA PREVENTION

#### MOUTHGUARD FABRICATION

#### ATHLETIC TEAM DENTISTS

Field Treatment Bag

#### SMOKELESS TOBACCO

Blood Pressure

Heart Rate

#### EATING DISORDERS

#### CONCLUSION

Sports dentistry is the treatment and prevention of orofacial athletic injuries and related oral diseases and manifestations.

The 1990 report of the Better Health Program entitled “Sports Injuries in Australia, Causes, Costs and Prevention” estimated that sports injuries cost Australia (population 18 million) about \$1.4 billion per year and that between 30% and 50% of these injuries are preventable. Multiply these numbers for the United States (population 296 million) and that would translate into \$23 billion per year. Participation in exercise and sports, whether positive or negative, will always remain a major consideration in the health of a national population.

In sports, the challenge is to maximize the benefits of participation and to limit injuries. Sports dentistry has a major role to play in this area. Prevention and adequate preparation are the key elements in minimizing injuries that occur in sports. Through sports dentistry, the prevention of orofacial trauma during sporting activities can be addressed. Included are teaching proper skills, such as tackling technique, purchase and maintenance of appropriate equipment, safe playing areas, and certainly the wearing and use of properly fitted protective equipment.

In some contact sports injury prevention, through properly fitted mouthguards, is considered essential,<sup>36</sup> e.g., football, boxing, martial arts, and hockey. Other sports, traditionally classified as theoretically noncontact sports—basketball, baseball, water polo, bicycle riding, roller blading, soccer, wrestling, racquetball, surfing, and skateboarding—also require properly fitted mouthguards because dental injuries unfortunately are a negative aspect of participation in these sports.

The National Youth Sports Safety Foundation (Needham, Mass.) reports several interesting statistics.<sup>17</sup> Dental injuries are the most common type of orofacial injuries sustained during participation in sports. Victims of dental avulsions who do not have their teeth properly preserved or immedi-



ately replanted will face lifetime dental costs estimated from \$10,000 to \$15,000 per tooth, the inconvenience of hours spent in the dental chair, and possibly other dental problems.

Treatment of orofacial injuries, simple or complex, is to include not only treatment of injuries at the dental office, but also treatment at the site of injury, such as a basketball court, football or rugby field, or swimming pool, where the dentist may not have the convenience of all the diagnostic tools available at an office. Knowledge and ability to do “on-site” differential diagnosis is essential.

Preseason orofacial screenings and examinations are essential in preventing injuries. Examinations should include health histories, at-risk dentitions, diagnosis of caries, maxillo-mandibular relationships, and the presence of orthodontic brackets and/or wires, loose teeth, dental habits, crown and bridge work, missing teeth, artificial teeth, and the possible need for any extractions. These extractions should be done months before playing competitive sports as to not interfere with competition or weaken jaws during competition. Determination of the need for a specific type and design of mouthguard is made at this time.

Mouthguard design and fabrication are extremely important. There are three types of mouthguards according to the Academy for Sports Dentistry, USA: stock, boil and bite, and custom made (vacuum or pressure laminated).\*

First of all, it is essential to educate the public that stock and boil and bite mouthguards bought at sporting good stores do not provide the optimum treatment expected by the athlete. These ill fitting mouthguards cannot deal with idiosyncrasies athletes and children may have. If everyone had the same dentition; were of the same gender; played the same sport under the same conditions; had the same experience and played the same position at the same level of competition; and were the same age and same size mouth, with the same number and shape of teeth, prescribing a standard mouthguard would be simple. This is the precise reason why mouthguards bought at sporting good stores, without the recommendation of a qualified dentist, should not be worn.

Idiosyncrasies are to be noted during mouthguard design and fabrication. These may include jaw relationships for which mouthguards may have to be designed on the mandibular arch such as a Class 3 prognathic bite. Otherwise, where possible, mouthguards should be built on the maxillary (upper) arch.

Erupting teeth (ages 6 to 12) should be noted so the mouthguard can be designed to allow for eruption during the season. Boil and bite mouthguards do not allow for this eruption space.

For patients with braces, special designs for the mouthguards are essential to allow for orthodontic movement without compromising on injury prevention and fit. This can only be achieved through consultations with a dentist.

Sports dentistry also includes the need for recognition and referral guidelines to the proper medical personnel for

nondental related injuries that may occur during a dental/facial injury. These injuries may include cerebral concussion, head and neck injuries, and drug use. We are NOT suggesting that dentists treat these injuries, but as health professionals dentists should be able to recognize these entities and refer these patients to the proper medical personnel. For example, if a patient comes into the office with a fractured or avulsed tooth, the dentist must rule out the possibility of a head injury or concussion before treating the patient for the dental injury (see Chapter 2). If certain symptoms are present, such as persistent headaches or nausea, immediate referral to medical personnel is essential.

Smokeless tobacco should also be included and addressed under sports dentistry. Smokeless tobacco is often associated with certain sports, and the public should be educated on the dangerous properties and consequences of using smokeless tobacco.

It is common for dentists to recognize the symptoms of anorexia and bulimia through dental examination. Eating disorders are more frequent than one may think in female athletes.\* Women’s gymnastics, volleyball, and basketball are just a few sports in which eating disorders have been observed. Erosion patterns in the teeth, caused by gastric acids, often help dentists in the differential diagnosis of eating disorders. These patients need to be referred to the proper medical and psychological health professional.

## ATHLETIC MOUTHGUARDS

### History of Mouthguards

In one paper<sup>44</sup> it was reported that the first resemblance of a mouth protector was documented in approximately 1892 by Dr. Woolf Krause, who practiced dentistry in London. He placed strips of gutta-percha over the upper incisors of a boxer requesting the patient to bite hard on this thus providing protection. These were not permanent because this was done approximately one half hour before entering the ring. Fighters believed to wear these primitive protectors were Jack Daniels and Pedlar Palmer. Later, his son, Dr. Philip Krause, who was also interested in boxing, and was a young fighter himself, made mouthguards from vela rubber for fighters. He claims to have been the first amateur fighter to wear permanent mouthpieces made in his father’s laboratory in London and later made mouthguards for Ted Kid Lewis before he turned professional.

In the United States, the first mouthpiece was probably constructed by Dr. Thomas A. Carlos from Chicago.<sup>44</sup> He claims to have made his first mouthguard from an impression in 1916. Later, in 1919, Dr. Allen Frankel, also a Chicago dentist, made mouthpieces for many of the world’s greatest fighters. Later American dentists Abrams, Jacobs, Hagely, Mayer, and Kerpel followed in the footsteps of the Krauses, Carlos, and Frankel. Their contributions were made in the

\*References 10, 16, 24, 26, 32, 34, 38-41, 45.

\*References 3, 7, 9, 30, 33, 49.

1920s and 1930s. By 1927, the mouthpiece was as much a part of a boxer's equipment as shoes and a foolproof cup.

Mouthguards have evolved as an accepted form of orofacial protection from the over-the-counter variety to professional custom-made types.

## TYPES OF ATHLETIC MOUTHGUARDS

Properly diagnosed, designed, and custom-fabricated mouthguards are essential in the prevention of athletic orofacial injuries.

In Dr. Raymond Flander's 1995 study,<sup>22</sup> he reported on the high incidence of injuries in sports other than football, in both male and female sporting activities. In football, in which mouthguards are worn, 0.07% of the injuries were orofacial. In basketball, in which mouthguards are not routinely worn, 34% of the injuries were orofacial. Various degrees of injury, from simple contusions and lacerations to avulsions and fractured jaws, are being reported.

It is estimated by the American Dental Association that mouthguards prevent approximately 200,000 injuries each year in high school and collegiate football alone.

A properly fitted mouthguard must be protective, comfortable, resilient, tear resistant, odorless, tasteless, not bulky, cause minimal interference to speaking and breathing, and (possibly the most important criteria) have excellent retention, fit, and sufficient thickness in critical areas.

Unfortunately, the word "mouthguard" is universal and generic, and includes a large range and variety of products, from over-the-counter models bought at the sporting goods stores to professionally manufactured and dentist-prescribed custom-made mouthguards. Presently, a vast majority of the mouthguards worn are of the variety that are purchased at sporting good stores.<sup>4</sup> The others are of the custom-made variety diagnosed and designed by a health professional (dentist and/or athletic trainer). There are three types of mouthguards presently available.

### Stock Mouthguards

The stock over-the-counter mouthguards, available at most sporting good stores, come in limited sizes (usually small, medium, and large) and are the least expensive and least protective (Fig. 11-1). The prices range from approximately \$5 to \$35. These protectors are ready to be used without any further preparation; simply remove from the package and immediately place in the mouth. They are bulky and lack any retention, and therefore must be held in place by constantly biting down. This interferes with speech and breathing, making the stock mouthguard the least acceptable and least protective. This type of mouthguard is often altered and cut by the athlete in an attempt to make it more comfortable, further reducing the protective properties of the mouthguard. It has been suggested and advised in the medical-dental literature that these types of mouthguards not be worn because of their lack of retention and protective prop-



A



B

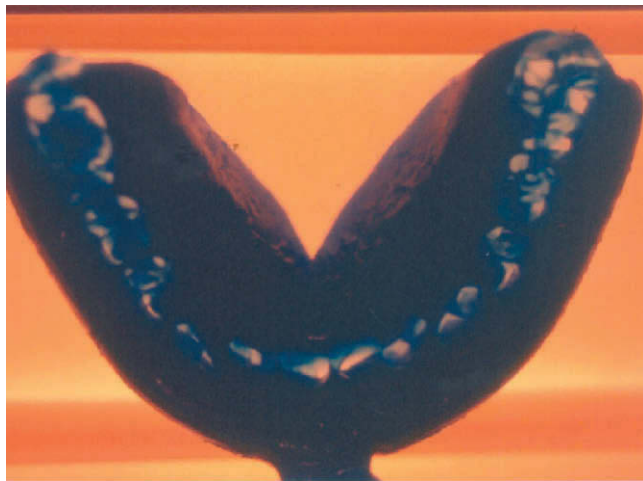
Figure 11-1 Type I stock mouthguards.

erties.\* Sports dentists and health professionals interested in injury prevention usually do not recommend this type of mouthguard to patients and athletic teams.

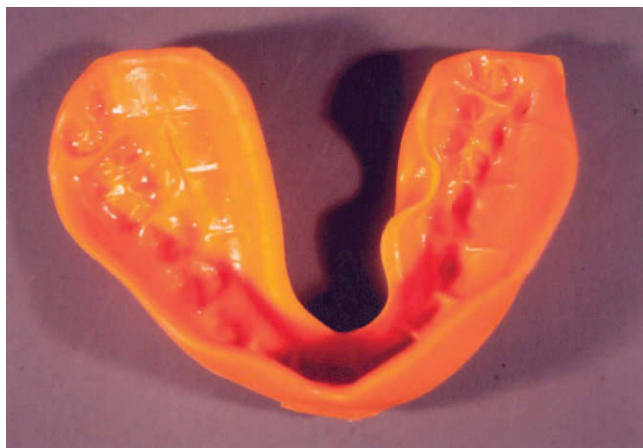
### Boil and Bite Mouthguards

Presently, this over-the-counter mouthguard is the most commonly used on the market because of convenience and price (Fig. 11-2). Most marketing and advertising campaigns in the past have been for this type of mouthguard. Made from thermoplastic material, it is immersed in boiling water

\*References 2, 16, 29, 32, 38-40, 51, 52.



A



B



C

**Figure 11-2** Type II boil and bite mouthguards.

and formed in the mouth by using finger, tongue, and biting pressure. Available in limited sizes, these mouthguards often lack proper extensions and typically do not cover all the posterior teeth. Because of large variances in dental arch lengths, many boil and bite mouthguards do not cover all posterior

teeth in high school and collegiate athletes. Athletes also cut and alter these bulky and ill fitting boil and bite mouthguards because of their poor fit, poor retention, and gagging effects. This in turn further reduces the protective properties of these mouthguards. When the athlete cuts the posterior borders or bites through the mouthguard during forming, the athlete increases the chance of injury, possibly concussion, from a blow to the chin. Some of these injuries, such as concussion, may cause lifelong effects. Proper thicknesses and extensions are necessary for proper mouthguard protection.

Park et al<sup>42</sup> at the First International Symposium on Biomaterials in August of 1993 reported that boil and bite mouthguards provide a false sense of protection because of the dramatic decrease in thickness occlusally during the molding and fabrication process. Park further stated that “unless dramatic improvements are made, they (boil and bite mouthguards) should *not* be promoted to patients as they are now.” He reported that boil and bite mouthguards decrease in occlusal thickness 70% to 99% during molding, thus taking away the protective properties of the mouthguard.

Care should be taken by the public when bombarded with clever marketing schemes, claims, and promotions by stock and boil and bite mouthguard companies. The bottom line is that stock and boil and bite mouthguards do not provide the expected care and injury prevention that a properly diagnosed and fabricated custom-made mouthguard does. Sports dentists and health professionals interested in injury prevention do not recommend store-bought boil and bite mouthguards to patients and athletic teams.\* The public deserves the best quality of care in injury prevention, and boil and bite mouthguards do not provide this quality.

### Custom-Made Mouthguards

Custom-made mouthguards are supplied by dentists. Custom mouthguards provide the dentist with the critical ability to address several important issues in the fitting of the mouthguard. Several questions must be answered before the custom mouthguard can be fabricated and must be addressed at the preseason screening or dental examinations (Box 11-1).

These are important questions that the sporting good store retailer and the boil and bite mouthguard cannot begin to address.

Custom-made mouthguards designed by the dentist are the most satisfactory of all types of mouth protectors. They fulfill all the criteria for adaptation, retention, comfort, and stability of material. They interfere the least with speaking and have virtually no effect on breathing. There are two categories of custom mouthguards, the *vacuum thermoformed mouthguard* and the *pressure laminated mouthguard*.

\*References 2, 16, 29, 32, 38-40, 51, 52.



**BOX 11-1****Necessary Information for Custom-Made Mouthguards**

- Is the mouthguard designed for the particular sport being played?
- Is the age of the athlete and the possibility of providing space for erupting teeth in mixed dentition (age 6 to 12) going to affect the mouthguard?
- Will the design of the mouthguard be appropriate for the level of competition being played?
- Does the patient have any history of previous dental injury or concussion, thus needing additional protection in any specific area?
- Is the athlete undergoing orthodontic treatment?
- Does the patient have dental caries and/or missing teeth?
- Is the athlete being helped by a dentist and/or athletic trainer or by a sporting goods retailer not trained in medical-dental issues?

**Vacuum Thermoformed Mouthguard**

The vacuum thermoformed mouthguard is made from a stone cast of the mouth, usually of the maxillary arch, using an impression and cast mold fabricated by the dentist. A thermoplastic mouthguard material is adapted over the cast with a special thermoforming vacuum machine.

There are a wide variety of vacuum machines available (Figs. 11-3, 11-4). The internal adaptation of the mouthguard may vary and subsequently the fit of the thermoformed appliances may vary depending on the amount of suction available, the strength of the motors, and the heating elements of the various devices. It is accepted that vacuum machines do not deliver the accuracy of the more precise pressure machines<sup>38</sup> (Figs. 11-5, 11-6).

The most common material for this use is a poly (ethylene vinyl acetate [EVA]) copolymer. After the vacuum mouthguard is formed, it is then trimmed and polished to allow for proper tooth and gingiva adaptation. All first molar teeth should be covered, muscle attachments unimpinged, and full vestibular coverage achieved for maximum retention. Vacuum machines are adequate for single layer mouthguards. However, it is now being shown in the dental literature that multiple-layer mouthguards (laboratory pressure laminated) may be preferred to the single-layer vacuum mouthguards.\* These vacuum custom mouthguards are still superior to the store-bought stock and boil and bite mouthguards because they have a better fit, made from a mold of one's mouth, and are designed by a dentist.

Strap attachments to helmets may be requested and are easily adapted to the custom-made mouthguard, although not necessary because of the good fit of the mouthguard.



**Figure 11-3** Conventional vacuum.

Custom-made mouthguards can be fabricated through the dental office or commercial laboratory for a nominal fee.

**Pressure Laminated Mouthguard**

A custom laboratory, multiple-layered pressure laminated mouthguard can be fabricated for full contact sports by laminating two or three layers of EVA material to achieve the necessary thickness. Lamination is defined as the layering of mouthguard material to achieve a defined end result and thickness under a high-heat and pressure environment. Efficient and complete lamination cannot be achieved using low heat and vacuum devices. The layers will not properly fuse together with most vacuum machines, but will chemically fuse under high heat and pressure with pressure machines (Fig. 11-7).

There are presently three pressure machines on the market. The Druformat from Dreve Dentamid in Unna, Germany; the Erkopress from Erkodent in Pfalzgrafenweiler, Germany; and the Biostar from Scheu Dental in Iserlohn, Germany. These machines are accepted as the state of the art for thermoforming, delivering the most precise adaptation and fit for appliances. The amount of pressure (up to 6 bars) and heat delivered far exceeds that of conventional vacuum

\*References 2, 16, 29, 32, 38-40, 51, 52.

70  
A



B

C



**Figure 11-4** Modern vacuums. **A**, Vacformat 2000. **B**, Erkoform-RVE. **C**, Vacformat U. (**A** and **C**, Courtesy Dreve-Dentamid GmbH, Germany; **B**, Courtesy Erkodent, Germany.)



**Figure 11-5** Type III custom-made vacuum with outdated conventional systems. Note poor internal adaptation.



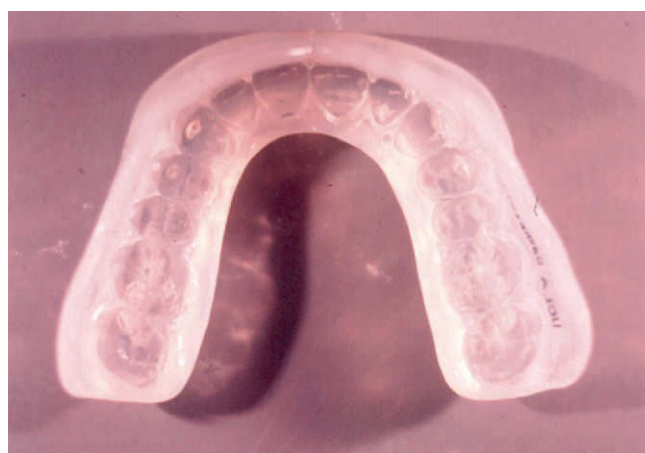
**Figure 11-6** Type III custom-made vacuum with modern vacuum systems. Note better internal adaptation.



A



B



C

**Figure 11-7** Type III custom-made pressure laminated mouthguards. Note uniform thickness and precise internal adaptation.

machines. These machines are not used solely for fabrication of athletic mouthguards, but also for many thermoforming appliances used every day by dentists, such as orthodontic retainers, model duplications, periodontal stents, implant stents, bleaching trays, custom-impression trays, bruxism

splints, matrix stents for provisional crown and bridge temporaries, antisnoring appliances, temporomandibular joint (TMJ) splints, copings for crowns, and partial denture and full denture repairs.

Protective thickness is important because as the thickness of the mouthguard material increases logarithmically, the transmitted impact force decreases logarithmically. Until recently, vacuum fabricated mouthguards have been the standard of care for protective mouthguards, despite the resulting mouthguards not fully adapting to the model because of the lesser amount of pressure and vacuum created by these machines.

Hunter<sup>32</sup> reported that mouthguards should be of a certain thickness, without being bulky. He suggests labial thickness of 3 mm, palatal thickness of 2 mm, and occlusal thickness of 3 mm. The mouthguard material should be bio-compatible and have good physical properties and last for at least 2 years. These are recommended thicknesses. It should be noted that each athlete should be evaluated individually for thickness and design to promote comfort and sufficient protection. According to Hunter, there are three advantages of pressure formed lamination (Box 11-2).

Therefore mouthguards must maintain minimal and consistent thicknesses in critical areas. These thicknesses may have to vary according to the athlete's individual needs for optimal protection. The thicker materials (3 to 4 mm) are more effective in absorbing impact energy and the thinner materials show notable deformation at the site of impact. These mouthguards are not bulky nor uncomfortable.

The clinician cannot expect that a 3-mm-thick material will remain 3-mm thick after fabrication. This is a physical impossibility because of shrinkage during fabrication adaptation. Vacuuming a commercially laminated 3-mm sheet of EVA will give the same unsatisfactory results. Therefore laboratory pressure lamination procedures must be used incorporating two or more EVA materials to achieve a desired end result of 3-mm to 4-mm thickness occlusally. This will allow the clinician to monitor and measure the results before delivery of these mouthguards.

#### BOX 11-2

#### Advantages of Pressure Formed Lamination Mouthguards

- Precise adaptation.
- Negligible deformation when worn for a period of time. The combination of the relatively high heat and pressure used in construction of laminated mouthguards means that the mouthguard material has virtually no elastic memory.
- The ability to thicken any area as required and place any inserts that may be needed for additional wearer protection.



There are presently two ways of obtaining a pressure laminated mouthguard: dentist fabrication with the Druformat, Erkopress-2004, or Biostar in the dental office or referral to a qualified commercial laboratory presently using the pressure lamination technique.

## ORTHODONTIC AND MIXED DENTITION CONSIDERATIONS

Mouthguards for orthodontic patients are the most critical and difficult mouthguards to fabricate. It is extremely important that the clinician consult with a pediatric dentist and/or an orthodontist for a successful result in the diagnosis, design, and fabrication of mouthguards. Many orthodontists do not want their patients wearing custom mouthguards because of the unfounded perception that these mouthguards will negatively interfere with the movement and completion of their orthodontic treatment. The orthodontist will often prescribe and/or provide stock or boil and bite mouthguards to these patients because these mouthguards have such a poor fit and presumably do not interfere with orthodontic tooth movement. These ill-fitting mouthguards also have the poorest compliance, resulting in soft tissue, fracture, and luxation injuries. Through cooperation, mouthguard designs can be fabricated so as to not interfere with the goals of the orthodontist while at the same time contributing to compliance and injury prevention. By slightly blocking out on fabrication models, mouthguards can be made to fit and provide the intended protection. These orthodontic mouthguards must be replaced regularly at the discretion of the dentists because the dentition is constantly changing.

In cases in which there is a mixed dentition, usually ages 6 to 12, blocking out the path of eruption of the adult dentition on the fabrication model is sufficient for proper fit and protection.

In both cases, an excellent model with full vestibular extensions is essential for fabrication because much of the retention will be tissue retention from the alveolar mucosa. It is also suggested that expiration dates be placed on these mouthguards to remind patients and parents that these need to be replaced on a regular basis, approximately every 4 to 6 months.

## COMPLIANCE AND ACCEPTANCE OF MOUTHGUARDS

At present, mouthguard compliance is poor.\* There is a perception that mouthguards are uncomfortable, do not fit, are bulky, and interfere with breathing and speaking. Ninety

percent of today's mouthguards worn are of the stock or boil and bite variety, and it is the perception by the public and coaches that these are the only available mouthguards. The majority of athletes are not wearing properly made dentally diagnosed and designed custom-made mouthguards provided by sports dentists. Through persistent education and more involvement at the dental school level, patients and athletes will be better informed on proper orofacial protection. Dentists and patients should realize that only through custom-made mouthguards can the highest level of protection and acceptance be achieved. If the mouthguard is comfortable and fit properly, the athlete will wear it. If it is uncomfortable and does not fit properly, the athlete will not be compliant. In sports in which straps are *not* applicable, such as basketball, rugby, soccer, water polo, and martial arts, there is a decline in acceptance because it is not common knowledge that custom mouthguard fabrication can provide comfort and protection. In sports in which straps are available due to helmet attachment, we see an increase in use. However, in these sports we often notice these mouthguards out of the mouth and dangling from the helmet. With properly fitted and custom-fabricated mouthguards, there is no need for straps. Comfort and protection are achieved and the mouthguards tend to stay in the mouth.

The literature describes the factors, such as fit, and comfort affect attitudes and compliance.\* But players have limited educational opportunities to learn about the effectiveness and types of mouthguards. Coaches, dentists, and health care providers should engage in more preventive educational programs to increase player attitudes and compliance.

Data suggest that the use of mouthguards in competition is not consistently enforced by athletic trainers, coaches, or game officials, and that mouthguards are not routinely worn by athletes.\* These results raise legitimate concerns for all physicians, athletic trainers, coaches, and governing bodies involved with sports. Investigators<sup>22</sup> reported in 2000 that despite knowledge of mouthguard protection, only five amateur sports and one professional sport have regulations requiring the use of mouthguards. Even in the sports that require their use, compliance is not universal. Attitudes of coaches, officials, parents, and players about wearing mouthguards influence their usage. Studies of the attitudes of these groups reveal that coaches are perceived as individuals with the most impact on whether or not players wear mouthguards.\* There is reluctance among college football officials to enforce mouthguard violations that they believe are inappropriate. Parents see themselves as having responsibility in determining mouthguard use; however, their views about when and for whom mouthguards are necessary reveal a lack of complete understanding of the

\*References 12, 19, 23, 24, 53, 54.

\*References 6, 12, 18, 19, 23, 24, 53, 54.

benefits. Resistance on the part of players stems from the physical characteristics of the mouthguard, interference with breathing and speech, and the effect on the players' image. Education on the effectiveness of properly fitted mouthguards for injury prevention, information on the risk for injury, more availability of comfortable and appealing custom-made mouthguards, and development of an approach for expanding regulations are all tools that can lead to the development of more positive attitudes and increased usage.

Concerning parental attitudes toward mouthguards,<sup>18</sup> investigators reported in 1997 that parents were asked questions such as "Who should be responsible for mouthguard wear?" "What sports should require mouthguards?" and "Has [their] child ever sustained an oral or facial injury?" The parental responses indicate that mouthguard enforcement is the responsibility of both parents and coaches. Of the total injuries reported, 19% were sustained in basketball, 17% in baseball, and 11% in soccer. Despite these high injury rates, however, there was a lack of perceived need for mouthguard use in these sports. When asked which sports should require a mouthguard rule, the sports that generated the most responses were, in decreasing order, football, boxing, ice hockey, wrestling, field hockey, and karate. Parents were more likely to require mouthguards for their sons than daughters, and more likely to require them for their children who participated in a mandatory mouthguard sport, a contact sport, or who had been previously injured. The authors conclude that because parents view themselves as equally responsible as coaches for maintaining mouthguard use, both groups should be targeted and approached as a possible source for the recommendation of mandatory mouthguard rules in basketball, baseball, and soccer.<sup>18</sup>

Through the above literature reviews, there is one common denominator: *education*. Until all concerned in the safety and well being of children and athletes are properly educated on the progress and modern status of mouthguards, those uninformed will continue to pursue the stock and boil and bite over-the-counter variety of mouth protection, thus lowering the acceptance and compliance of athletic mouthguards. Once these parents, coaches, game officials, physicians, athletic trainers, and governing bodies involved with sports are properly informed and educated, then compliance will increase due to the proper fit and comfort of custom-made mouthguards.

## ROLE OF THE DENTIST IN OROFACIAL TRAUMA PREVENTION

The dentist's role in trauma prevention involves patient education, mouthguard assessment and design (Box 11-3), and mouthguard fabrication.

### BOX 11-3

#### Determining Factors for Mouthguard Design

- Age
- Sport played
- Level of competition
- Past history of dental trauma

### BOX 11-4

#### Steps of Fabrication of Custom-Made Mouthguards

1. Impression
2. Fabrication
3. Trimming and polishing
4. Placement and occlusal equilibration

The age of the patient-athlete dictates design considerations as a result of dentition alterations. When the patient-athlete first considers competition, he or she is sometimes in the developmental ages. This would involve mixed dentition and or orthodontic considerations. Mouthguards should be designed and replaced accordingly. During developmental ages, mouthguards should be replaced every 4 to 6 months. Once full development has been achieved, replacement is only necessary when dictated by loss, changes in dentition because of dental treatment, and wear causing loss in retention, thickness, and comfort.

The sport played and levels of competition also dictate mouthguard design (Fig. 11-8). The more aggressive the sport, the more aggressive the mouthguard design. Aggressiveness is addressed by increased thickness of material, and by the introduction of soft or hard inserts between laminated layers. Past history of trauma is also important in design. The literature reports that more than 80% of orofacial hard tissue injuries occur to the front four maxillary incisors.<sup>24,35,46</sup> Thus if there is a previous history of trauma to these anterior teeth and/or previous endodontic and restorative treatments rendered (resin bonding, veneers, or crowns), design considerations are strongly suggested to increase the thickness of material and/or introduce soft or hard inserts in between laminated layers.

## MOUTHGUARD FABRICATION

There are two options for Type III mouthguard fabrication: self-fabrication in the office or referral to a qualified dental laboratory (Box 11-4). Which option to take is dependent on both the clinician's ability and the equipment that is available for use.



Figure 11-8 Various mouthguard designs.



A



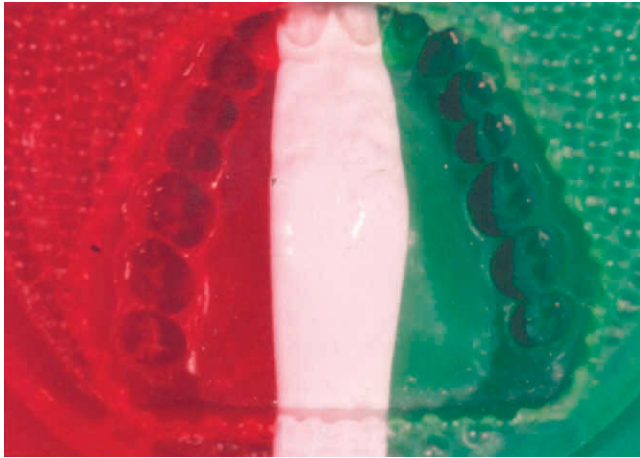
B

Figure 11-9 A, Mouthguard created with conventional vacuum internal adaptation (B).

There is a big difference in the internal adaptation of the mouthguard when comparing the older conventional vacuum machine to the newer pressure machines (Figs. 11-9, 11-10). One can easily see the adaptation from their machine by making a mold from a 3-mm sheet of EVA material. Take

out the original model and pour stone into EVA material. The result will show you the adaptation of your machine (Figs. 11-11 to 11-14). From there, decisions can be made on purchasing the newer varieties of vacuum machines or investing in the state of the art pressure machines.





A



B

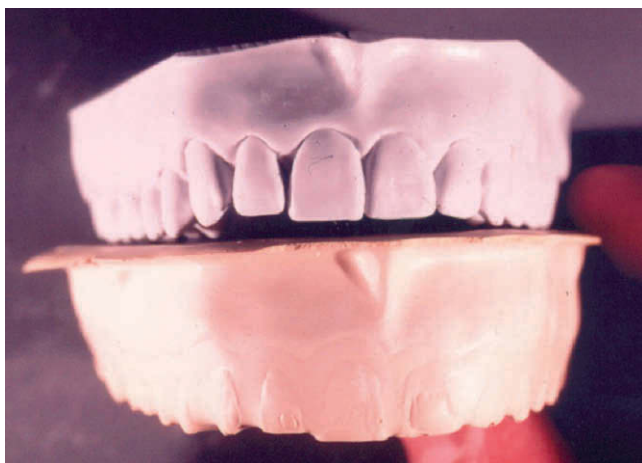


C

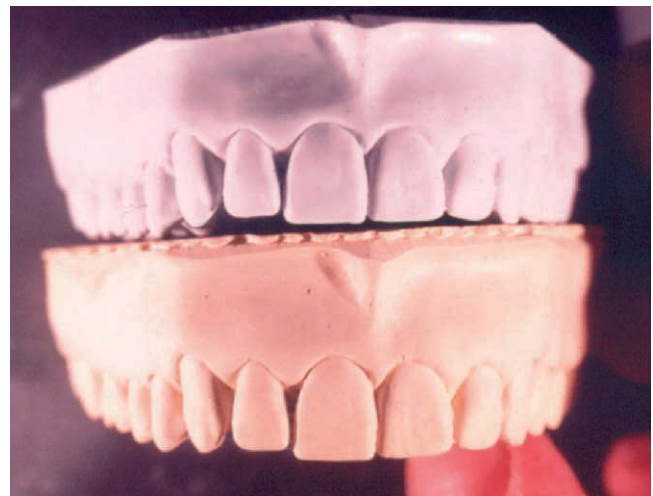


D

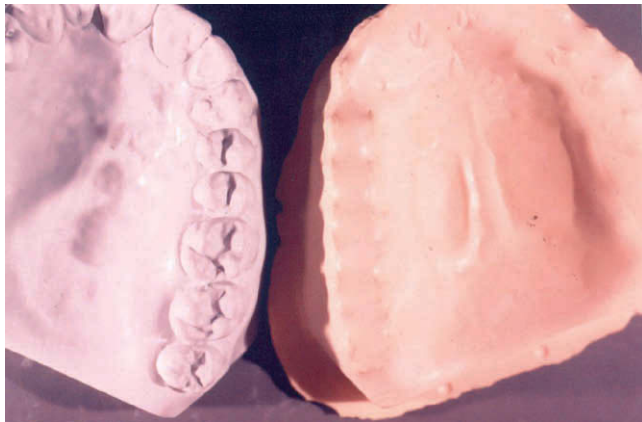
**Figure 11-10** A, Mouthguard created with pressure internal adaptation with state of the art pressure machines. B, Drufoformat TE. C, Erkopress ES-200 E. D, Biostar. (B Courtesy Dreve-Dentamid GmbH, Germany; C courtesy Erkodent, Germany; D courtesy Great Lakes Orthodontics, Tonawanda, N.Y.)



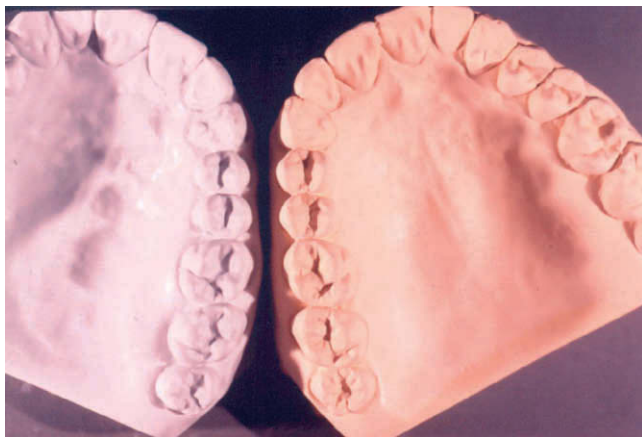
**Figure 11-11** Original and vacuum model.



**Figure 11-12** Original and pressure model.



**Figure 11-13** Original and vacuum model.



**Figure 11-14** Original and pressure model.

The impression is critical to the end result (Figs. 11-15 to 11-19). Similar to any restorative procedure requiring an impression, the better the impression, the better the appliance. It has been the author's experience that an excellent method for impressions for mouthguard appliances is the Accu-Dent System II. Accu-Dent multicolloid impression systems eliminate the need for custom impression trays, allowing the dentist to create master casts in a more efficient and profitable manner. The impression gels are formulated in two viscosities: a light bodied Syringe Gel that captures soft tissues and a heavy body Tray Gel for hard tissue. Ideal for athletic custom-made mouthguards, the Accu-Dent System II can also be used for partials, immediate dentures, orthodontics, and splints. The light bodied Syringe Gel is applied with a special tip to capture detail while eliminating air bubbles.

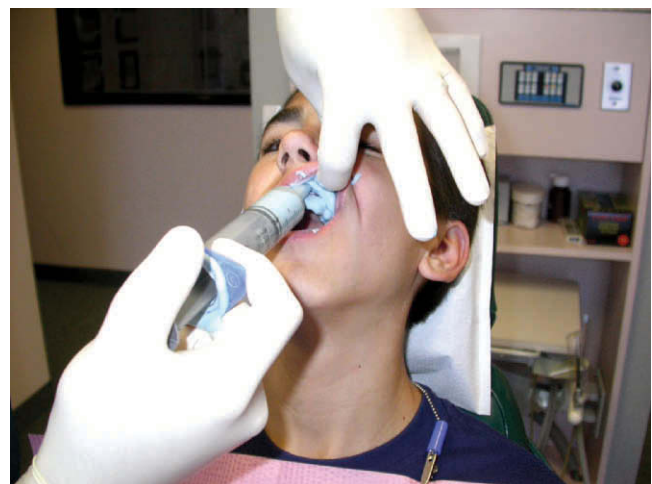
Once the impression has been taken, immediate pour-up with a hard die stone is recommended (Fig. 11-20). Care is taken to capture all vestibular borders. A large base is not necessary because it will be taken off while trimming the model.



**Figure 11-15** Impression setup.

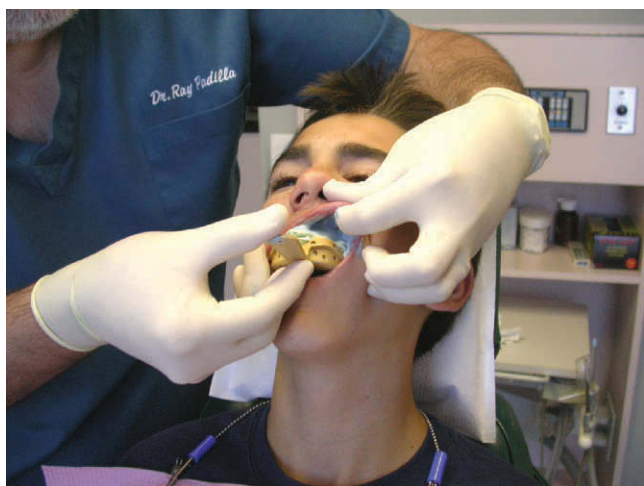


**Figure 11-16** Light body application to teeth.



**Figure 11-17** Light body application to vestibular areas.





**Figure 11-18** Heavy body seat with tray.



**Figure 11-21** Mark the vestibular margin outline.



**Figure 11-19** Accudent final impression.



**Figure 11-22** Model trimming to vestibular margins.



**Figure 11-20** Die stone model.

After the model has become hard and set, usually about 45 minutes, mark the highest margins of the vestibular border with pencil for reference during trimming (Figs. 11-21 to 11-23). Trim the excess stone carefully to these borders. By including these vestibular borders, the mouth-guard will have more retention because of increased surface adaptation and will also help protect the alveolar bone from further trauma.

Once the model has been properly trimmed and dried, it must be lubricated to allow easy separation after fabrication (Fig. 11-24). The author's lubricant of choice is orthodontic model soap. Soak the model in the soap for approximately 1 hour, then dry and polish with a dry towel. The model is now lubricated for the life of the model.

The model is now ready for the fabrication of the mouth-guard by pressure lamination using pressure machines. Note, this process is *not* performed in a (suck down) vacuum. It is positive pressure. There are presently three pressure





**Figure 11-23** Model trimming maintaining vestibular borders.



**Figure 11-24** Soak model in ortho model soap.

machines available for this process and all must be connected to a compressor for air pressure. They are the Druformat (Fig. 11-25) (by Dreve, Unna, Germany, distributed by Raintree Essix, LLC, Metairie, La.), the Erkopress (see Fig. 11-10) (by Erkodent, Pfalzgrafenweiler, Germany, distributed by Glidewell Laboratories, Newport Beach, Calif.), and the Biostar (see Fig. 11-10) (by Scheu Dental, Iserlohn, Germany, distributed by Great Lakes Orthodontics, Ltd, Tonawanda, N.Y.). For the purposes of this demonstration, the Druformat will be used.

A 3-mm to 4-mm-thick mouthguard will be made. Two layers of 3-mm EVA will be laminated together. It is important that this process be done in two separate steps to allow for proper thickness in the incisal and occlusal surfaces. If



**Figure 11-25** Druformat pressure thermoforming machine.

done in only one step, the thickness in these critical incisal and occlusal areas will be compromised. There is approximately 30% to 40% shrinkage of EVA material during fabrication, so two 3-mm sheets laminated together will form a 3-mm to 4-mm mouthguard.

After the heater switch and machine power are turned on, a 3-mm sheet of EVA is placed in the disc positioning ring (Figs. 11-26, 11-27). The trimmed model with marked extensions is placed on the tray table slightly off center toward the lingual.



**Figure 11-26** A sheet of EVA is placed in the disc positioning ring.



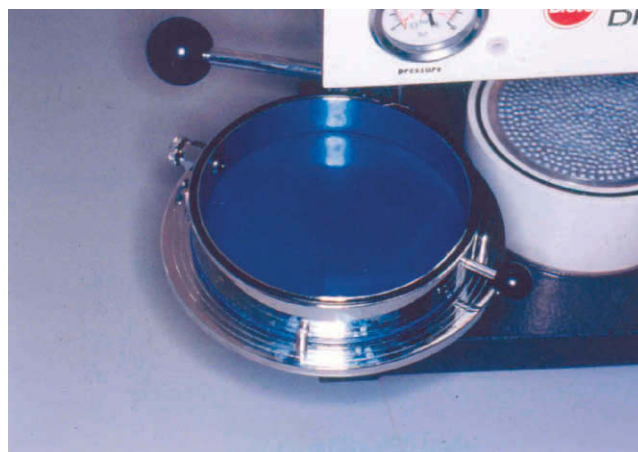
**Figure 11-27** A trimmed model is placed slightly off center toward the lingual.

A trimmed model is placed slightly off center toward the lingual (see Fig. 11-27).

The clamping ring is placed over the EVA sheet to lock it into position (Fig. 11-28).

The heater is placed into position over the model, allowing the EVA material to heat and soften to formable consistency (Figs. 11-29, 11-30).

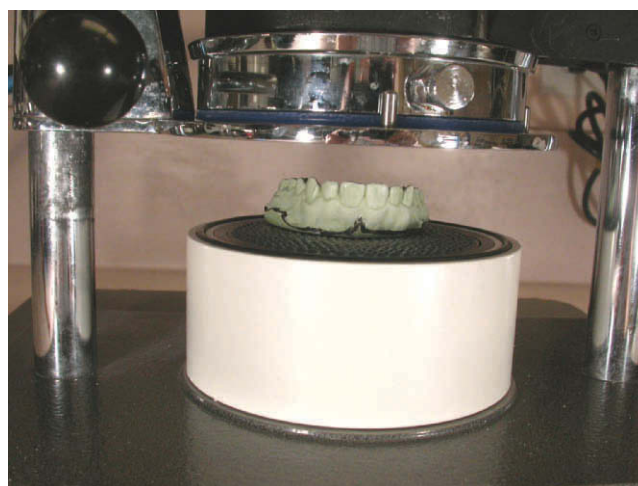
As the EVA material softens, it will begin to slump until it is touching the model (Fig. 11-31). At this stage, the first layer is ready to be thermoformed by pressure.



**Figure 11-28** A clamping ring locks the EVA sheet into place.



**Figure 11-29** Heating element in position over the model allowing the EVA material to heat up.



**Figure 11-30** Close-up of proper position of heating element.





**Figure 11-31** EVA material begins to slump until it touches model.



**Figure 11-33** A proper cool down period of 10 minutes prevents distortion.



**Figure 11-32** Pressure is used on the first layer and timing is critical to maintain pressure while cooling.



**Figure 11-34** White button on side panel is pressed until pressure indicator light is turned off.

On the upper left side of the Drufomat is a white button that activates the pressure (Fig. 11-32). This button must be pressed at the same time that the heater is removed from the EVA material. The pressure chamber will drop over the model and thermoform and pressurize the EVA to the model. A light will illuminate, signaling that the thermo process has begun, and the hands can be removed from the machine. If the hands are removed before the light activation, the pressure will not be maintained.

The EVA material must now cool for a minimum of 10 minutes (Fig. 11-33). The EVA material should not be manipulated and removed from the pressure chamber until it has completely cooled to prevent any distortion.

Once the time has elapsed, the white button is depressed until the pressure indicator light turns off, releasing the pressure in the chamber (Figs. 11-34, 11-35). The heater lever is



**Figure 11-35** The heater lever is slowly pushed toward the cylinder until the cylinder rises.





**Figure 11-36** Completed pressurized and cooled first layer.



**Figure 11-37** Essix electric hot knife.

slowly pushed toward the cylinder. The pressure cylinder will rise.

The first layer is now completed (Fig. 11-36). It may be removed from the disc positioning ring and allowed to cool to room temperature before trimming to prevent distortion.

Once the EVA material has cooled, excess material may be trimmed off using a hot knife (Figs. 11-37 to 11-39). Care should be taken not to trim excessively. The lingual borders are trimmed 1 mm from the teeth, and the labial borders follow the penciled mucosal borders. The distal of the first molar is the minimal extension.

The first layer is now ready for identification labels and logos (Figs. 11-41 to 11-44). Any label machine may be used as long as it provides a small font (10 point maximum).



**Figure 11-38** Careful trimming of the first EVA layer to the desired dimensions.



**Figure 11-39** Care is taken not to trim excessively. Keep all labeled borders and extensions intact.



Figure 11-40 Full vestibular borders are maintained.



Figure 11-41 Lingual borders trimmed to within 1 mm of teeth.



Figure 11-42 Identification labeler.

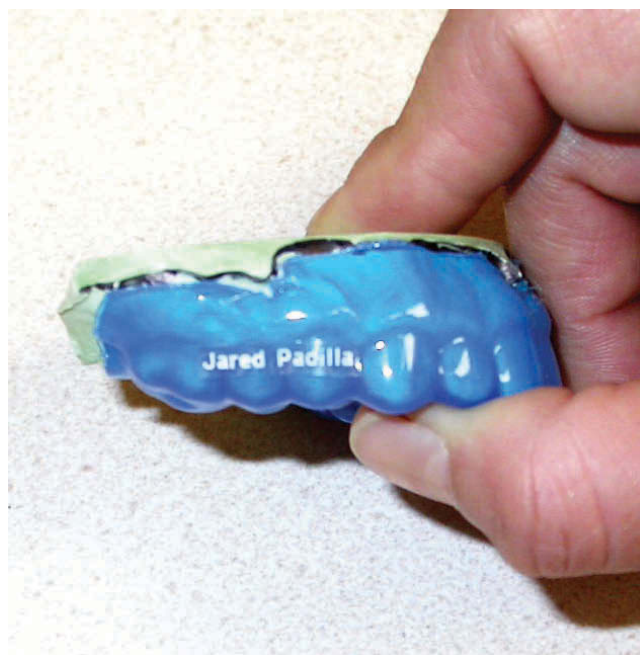


Figure 11-43 Patient's name identified on mouthguard.



Figure 11-44 Preparation for second layer placement of EVA material. Note the second layer is clear.

The mouthguard is now ready for the second layer, which will be laminated (Figs. 11-44 to 11-46). A clear sheet of EVA of desired thickness (in this case 3 mm) is placed in the disc positioning ring. The model with the first trimmed and labeled layer is placed on the positioning tray slightly off center toward the lingual. At this point, steps 29 through 37 (see Figs. 11-28 to 11-36) are repeated. The clear second layer begins to melt to the desired formable consistency. It is very critical that the second layer be allowed to become





**Figure 11-45** Second layer EVA material heats for thermoforming.



**Figure 11-46** Second clear layer EVA must be sufficiently hot for a successful lamination to first blue layer.

hot enough to predictably laminate to the first layer. It must heavily droop over the first layer. If not allowed to heat sufficiently, complete lamination will not occur and separation will take place in time. The pressure chamber is activated as in step 33 (see Figure 11-32), and the model allowed to cool under pressure for 15 minutes. Steps 34 to 36 (see Figures 11-33 to 11-35) are then repeated.

The clamping ring is removed and the laminated mouthguard is allowed to cool to room temperature to eliminate any chance of distortion, thereby ensuring a perfect tight fit (Fig. 11-47).



**Figure 11-47** With the clamping ring removed, the laminated mouthguard is cooled to room temperature before trimming to avoid distortion.

The second layer of excess EVA material is trimmed to proper extensions (Fig. 11-48).

The internal lingual extensions should be checked and marked with a pen to 1 mm from the teeth (Fig. 11-49). With a Dedico stone acrylic bur, the excess material is trimmed lingually to the marked extensions.

The mouthguard is then placed back on the model, and the margins are feather finished for comfort lingually, buccally, and labially. Any interference with muscle attachments should be removed (Figs. 11-50, 11-51).

With Essix Scotch wheels, the mouthguard is further trimmed and smoothed to desired thickness. All frenum attachments are relieved (Figs. 11-52, 11-53).

Final finish and polish are placed with wax remover (Fig. 11-54).

The 4-mm custom-made pressure laminated mouthguard is now completed (Figs. 11-55 to 11-58).

It is now important to try the mouthguard in the patient's mouth and check for fit and comfort (Figs. 11-59 to 11-62). Minimal adjusting may be necessary just as any other dental appliance insertion appointment. It is extremely important that a balanced occlusion be present. This is done by slightly warming the posterior occlusal surface of the mouthguard, taking extreme care not to overheat and distort, and placing the mouthguard in the patient's mouth and asking them to bite down very lightly and carefully until all posterior teeth occlude. Care should be taken not to bite down excessively





**Figure 11-48** Trimming the excess second layer.



**Figure 11-49** Internal lingual margins are trimmed to within 1 mm of teeth with stone acrylic bur.

as the occlusal separation of 3 mm to 4 mm must be maintained to ensure proper absorption of impact energy.

## ATHLETIC TEAM DENTISTS

Sports dentists are fast becoming a recognized part of the sports medicine team. This team usually consists of athletic trainers, orthopedic surgeons, internal medicine specialists,



**Figure 11-50** Margins feather finished for patient comfort.

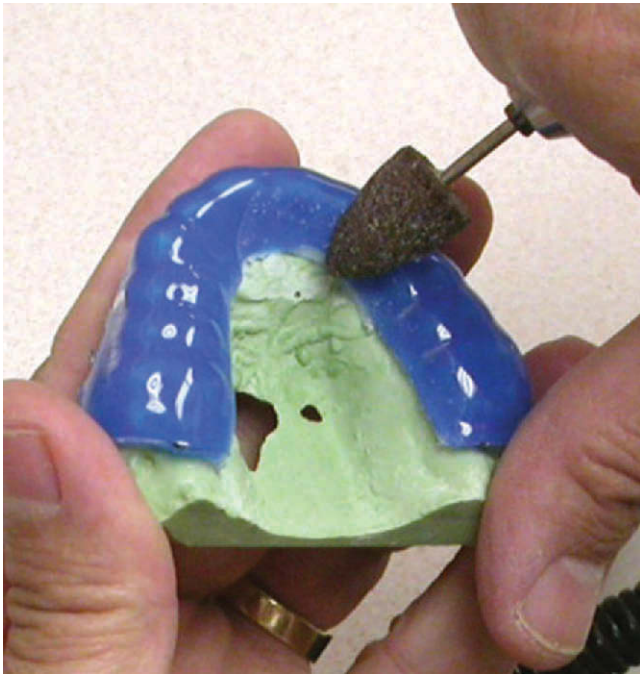
dentists, physical therapists, podiatrists, dermatologists, ophthalmologists, massage therapists, nutritionists, and chiropractors. Other medical specialties are also consulted on a regular basis.

Why the need for sports dentistry? Prevention and preparation are the key elements in minimizing injury. They allow the athlete to compete at their greatest levels, minimizing the potential of discomforts of oral injury and pain. Also, when athletic injuries occur, the sports trauma dentist is there to minimize injury and treat the athletes in a timely manner.

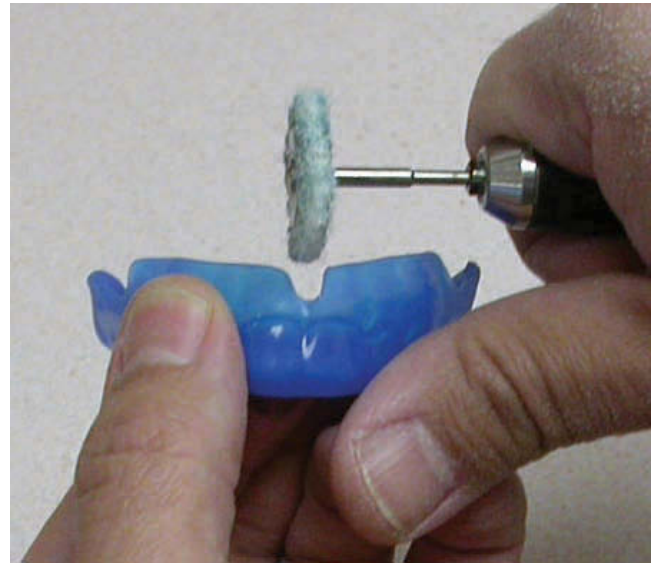
This is extremely important, especially with luxation and avulsion injuries for which the prognosis is dependent on how soon the athlete is treated after the injury. Time is of the essence and the injured athlete must be immediately seen and treated after the injury. Many professional and collegiate

teams have team dentists in attendance at athletic events where injuries may occur. The team dentist has many responsibilities (Box 11-5).

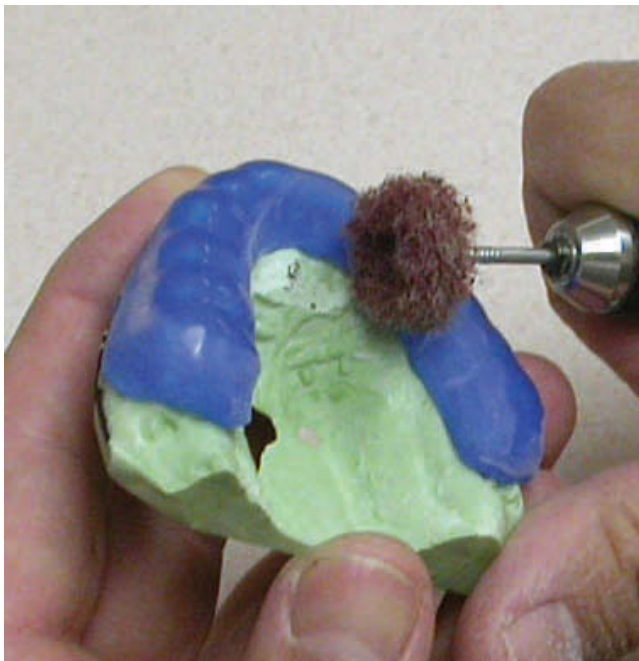
Preseason examinations are critical to the well being of the athletes. Through these exams, athletes may avoid



**Figure 11-51** Lingual smooth feather finishing is critical for patient comfort and acceptance.



**Figure 11-53** Final trimming of muscle frenum attachments.



**Figure 11-52** Final trimming with scotch wheel.





**Figure 11-54** Final polish with laboratory wax solvent.



**Figure 11-57** Lingual margins are thin, smooth, and polished for patient comfort.



**Figure 11-55** Laboratory completed pressure laminated mouthguard.



**Figure 11-58** Internal adaptation and completed extensions.



**Figure 11-56** Note that the full vestibular extensions were maintained throughout the trimming and polishing procedure.



**Figure 11-59** Tray setup.





**Figure 11-60** Preparing to fit the completed mouthguard in patient's mouth.



**Figure 11-61** Occlusal surface slightly warmed with butane.



**Figure 11-62** Completed mouthguard in balanced occlusion.

### BOX 11-5

### Responsibilities of a Team Dentist

- Possess the necessary didactic and clinical skills that will provide comprehensive orofacial care relating to the diagnosis and management of traumatic athletic injuries and reconstructive cosmetic restorative treatment.
- Provide dental first aid, including hard and soft tissue management.
- Provide dental services for athletes who need and require certain time restraints. For example, many elite athletes are training daily and need preventive and emergency treatment done in a timely manner as to not interfere with their academic and travel schedule.
- Provide quality assurance protocols, risk management, and on-field and locker room etiquette.
- Be informed on all banned substances (drugs) in athletics.
- Respect the athlete's privacy and confidentiality when dealing with the press and others.
- Have the ability to make on-field clinical judgments as to continuance of play without the clinical tools we are accustomed to in our offices.
- Assemble a dental field bag.
- Provide custom made athletic mouthguards.
- Be available for trauma care whether present at games or on call.
- Provide preseason dental screenings and examinations.

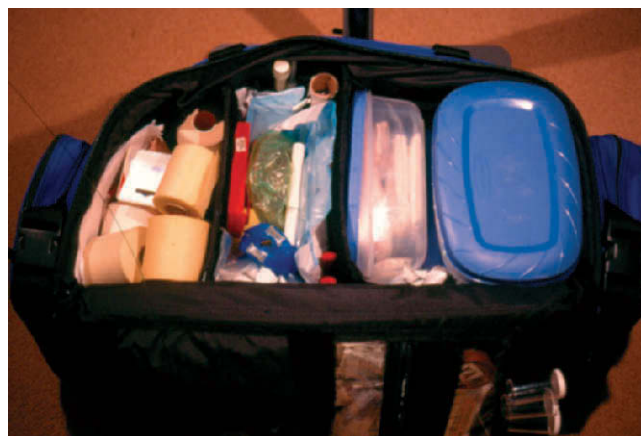
unexpected problems during the season. Athletes may be treated during the off season so they are not in time-consuming dental procedures during the season, which affects their practice and game time. This allows full concentration on the sport without dental complications. This also provides an opportunity for dental impressions for the fabrication of protective athletic mouthguards.

### FIELD TREATMENT BAG

The on-field dental emergency bag (Fig. 11-63) is an important part of the sports dentist's armamentarium (Box 11-6). These kits will vary according to the practitioner and the scope of treatment they are comfortable rendering on the field or in the locker room.



A



B

**Figure 11-63** A, Field bag. B, Contents of field bag.

#### BOX 11-6

#### Examples of Field Treatment Bag Contents

- Gloves
- Penlight
- Wound care kits
- Suture kit
- Anesthesia
- Variety of dental tools
- Antibiotic rinses
- Blood pressure cuffs
- Syringes, needles
- Betadine
- Tincture of benzoin
- Antibiotic ointment
- Sterile gauze
- Steri-Strips
- Butterfly closure bandages
- Intermediate restorative material (IRM)
- Glass ionomer restorative material
- Variety of permanent and temporary cements
- Bonding kits for avulsions and luxations consisting of etch, bonding agent, composite resin, and splinting material
- Portable curing light for bonding

#### BOX 11-7

#### Health Consequences of Smokeless Tobacco

- Dental caries and periodontal effects
- Oral cavity soft tissue alterations
- Leukoplakia (white, potentially precancerous intraoral lesions)
- Cancer of the oral pharynx and oral cavity
- Addiction from the nicotine
- Hemodynamic alterations due to the sugar content, leading to increased blood sugar levels

## SMOKELESS TOBACCO

In 1986, the U.S. Surgeon General concluded that the use of spit tobacco is not a safe substitute for smoking cigarettes or cigars because these products can cause various cancers and noncancerous oral conditions, and can lead to nicotine addiction (Box 11-7).<sup>5,55</sup> The tobacco industry has taken

advantage by exploiting the influence that athletes have on our young athletes and children. They are sponsoring sporting events such as professional rodeos, women's tennis, monster truck rallies, and car racing events, not to mention the association of baseball players with smokeless tobacco.

The addictive properties of nicotine have been recorded throughout the medical-dental literature.<sup>5</sup> Also the association of smokeless tobacco and cancer is well documented.<sup>1</sup> The U.S. Department of Health and Human Services reports that tobacco snuff contains the organic carcinogen nitrosonornicotine (NNN), which readily produces malignant tumors of the trachea, esophagus, liver, and oral cavity.<sup>5</sup> Research has shown that 15% to 20% of all adolescent men use smokeless tobacco. About half of these men have precancerous lesions in their mouth, and the chance of oral cancer developing in these individuals is 400% greater than in nonusers (Fig. 11-64).

Some athletes believe that smokeless tobacco enhances athletic performance. However, there is no evidence that smokeless tobacco gives athletes a competitive edge in reaction time, movement time, or total response. In fact, studies



A



B

**Figure 11-64 A**, The National Spit Tobacco Education Program (NSTEP) educates sports teams and the general public about the dangers of smokeless or spit tobacco. **B**, Carcinoma from smokeless tobacco. (Courtesy NSTEP, <http://www.nstep.org>.)

have shown a reduction in overall stamina because of an increase in heart rate and blood pressure.<sup>20,21</sup>

## BLOOD PRESSURE

Salt levels often are high in smokeless tobacco, which may be a factor for those with hypertension.<sup>25</sup>

## HEART RATE

Nicotine has ingredients that may increase the heart rate or cause blood clots. Heart disease, heart attacks, and strokes are all risks associated with tobacco use.<sup>31</sup>

Health professionals are actively trying to help patients realize that smokeless tobacco is *not* a safe alternative to smoking. Treatment of nicotine addiction is not an easy pro-

position. Smokeless tobacco has been banned by all youth baseball leagues, the National Collegiate Athletic Association, and all of Major League Baseball's minor league clubs. The Professional Baseball Athletic Trainers Association also discourages the use of smokeless tobacco.<sup>14</sup> Unfortunately, smokeless tobacco has not yet been banned from the American or National baseball leagues although the leagues do cooperate with the National Spit Tobacco Education Program to discourage children from using tobacco.

## EATING DISORDERS

The sports dentist may indeed be the first health professional to diagnose bulimia in athletes. Bulimia, which is more common than anorexia, describes a cycle of binge eating and purging.\*

Bulimia can begin when restrictive diets fail, or when the athlete has feelings of hunger associated with reduced calorie intake. In response to the binge, the athlete may feel guilty and purge by vomiting or may take laxatives, diet pills, or drugs to reduce fluids. Athletes may alternate between periods of bingeing and calorie restriction.

Signs and symptoms of bulimia include excessive weight loss or gain, being overly concerned with one's weight, visiting the bathroom immediately after meals, depression, and excessive dieting, followed by binge eating and always criticizing one's body. Women's gymnastics, volleyball, and basketball are just a few sports in which eating disorders have been documented; however men's sports may also be involved, such as gymnastics and wrestling.

During routine preseason screenings, dental erosion may be observed in these athletes. Dental erosion is described as progressive loss of hard dental tissue caused by a chemical process and not by bacterial action. Dental erosion is caused by the acidic content of the purged food coming in contact with the teeth (Fig. 11-65). This is an important diagnosis so that timely preventive and treatment measures may be implemented. Also, early proper referral to medical personnel is critical.

Other health complications of bulimia include lacerations of the oral cavity (injury due to self-induced vomiting), esophageal inflammation (acid from vomiting irritating the esophagus), esophageal tears and ruptures (force from vomiting may cause the tears in the esophagus), cardiac arrest, dehydration, and electrolyte imbalance.

## CONCLUSION

Orofacial trauma is not completely preventable, but measures can be taken to minimize the extent of injuries. Proper custom-fabricated mouthguards are the best defense against

\*References 1, 9, 28, 37, 48, 49.





A



B

**Figure 11-65** **A**, Facial view of dental erosion caused by acidic content of purged food. **B**, Palatal view. (Courtesy Dr. Helen Cornwell, Newcastle, Australia.)

sports-related injuries to the teeth. In the unfortunate event that a dental injury does occur, a thorough understanding of the assessment and management of these injuries is imperative. This will minimize any additional injury and maximize the prognosis. Orofacial trauma sustained from smokeless tobacco and eating disorders can be preventable. The dentist is often the first to recognize these behaviors. However, managing addictive behaviors and psychological disorders can be very challenging and often requires the assistance of individuals that specialize in these behavior patterns.

## REFERENCES

1. American Cancer Society website: [http://www.cancer.org/docroot/PED/content/PED\\_10\\_2x\\_Smokeless\\_Tobacco\\_and\\_Cancer.asp?sitearea=WHO](http://www.cancer.org/docroot/PED/content/PED_10_2x_Smokeless_Tobacco_and_Cancer.asp?sitearea=WHO)
2. Banky J, McCrory PR: Mouthguard use in Australian football, *J Sci Med Sport* 2(1):20-29, 1999.
3. Barron R, Carmichael R, Marcon M et al: Dental erosion in gastroesophageal reflux disease, *J Can Dent Assoc* 69(2):84-89, 2003.
4. Bemelmans P, Pfeiffer P: Incidence of dental, mouth, and jaw injuries and the efficacy of mouthguards in top ranking athletes, *Sportverletz Sportschaden* 14(4):139-143, 2000.
5. Berrettini WH, Lerman CE: Pharmacotherapy and pharmacogenetics of nicotine dependence, *Am J Psychiatry* 162(8):1441-1451, 2005.
6. Bolhuis JHA, Jeurs JMM, Floger GE: Dental and facial injuries in international field hockey, *Br J Sports Med* 21(4):174-177, 1987.
7. Bouquot JE, Seime RJ: Bulimia nervosa: Dental perspectives, *Pract Pesth Dent* 9:655-664, 1997.
8. Brisbane schoolboy swallows mouthguard, *Brisbane Australia Sunday Mail* Article 25/06/95.
9. Byrne S, McLean N: Eating disorders in athletes: a review of the literature, *J Sci Med Sport* 4(2):145-159, 2001.
10. Chaconas SJ, Caputo AA, Bakke NK: A comparison of athletic mouthguard materials, *Am J Sports Med* 13(3):193-197, 1985.
11. Chapman PJ: Concussion in contact sports and importance of mouthguards in protection, *Aust J Sci Med Sport* March, pp. 23-27, 1995.
12. Chapman PJ, Nasser BP: Attitudes to mouthguards and prevalence of orofacial injuries in four teams competing at the second world cup, *Br J Sports Med* 27(3):197-199, 1993.
13. Clegg JH: Letters to the editor, *Br Dent J* Oct 4, 1966.
14. Connolly GN, Orleans CT, Blum A: Snuffing tobacco out of sport, *Am J Public Health* 82(3):351-353, 1992.
15. Davis GT, Knott SC: Dental trauma in Australia, *Aust Dent J* 29(4):217-231, 1984.
16. Dennis C, Parker DAS: Mouthguards in Australian Sport, *Aust Dent J* pp. 226-235, June 1972.
17. Dental injury fact sheet, Needham, Massachusetts, 1992, National Youth Sports Foundation for the Prevention of Athletic Injury Inc.
18. Diab N, Mourino AP: Parental attitudes toward mouthguards, *Pediatr Dent* 19(8):455-460, 1997.
19. Duda M: Which athletes should wear mouthguards? *Physician Sports Med* 15(9), September 1987.
20. Edwards JG: An unusual case of nicotine dependence, *Psychol Med* 17(3):779-781, 1987.
21. Edwards SW, Glover ED: Snuff and neuromuscular performance, *Am J Public Health* 76(2):206, 1986.
22. Flanders RA, Mohandas B: The incidence of orofacial injuries in sports: a pilot study in Illinois, *JADA* 126:491-496, 1995.
23. Gardiner DM, Ranalli DN: Attitudinal factors influencing mouthguard utilization, *Dent Clin North Am* 44(1):53-65, 2000.
24. Gabris K, Tarjan I, Rozsa N: Dental trauma in children presenting for treatment at the department of dentistry for children and orthodontics, Budapest, 1985-1999, *Dent Traumatol* 17(3):103-108, 2001.
25. Hampson NB: Smokeless is not saltless, *N Engl J Med* 4:312(14):919-920, 1985.
26. Harmer PA: Protective equipment in womens lacrosse, *Sport Health Aust* 11(3):14-17.
27. Heintz WD: Mouth protection in sports, *Physician Sportsmedicine*, 7(2), Feb 1979.
28. Hobart JA, Smucker DR: The female athlete triad, *Am Fam Physician* 1;61(11):3357-3364, 3367, 2000.
29. Hoffmann J, Alfter G, Rudolph NK et al: Experimental comparative study of various mouthguards, *Endod Dent Traumatol* 15(4):157-163, 1999.
30. Hopkinson RA, Lock J: Athletics, perfectionism, and disordered eating, *Eat Weight Disord* 9(2):99-106, 2004.
31. Huhtasaari F, Lundberg V, Eliasson M et al: Smokeless tobacco as a possible risk factor for myocardial infarction: a population-based study in middle-aged men, *J Am Coll Cardiol* 15;34(6):1784-1790, 1999.
32. Hunter K: Modern mouthguards, *Dent Health Res Found Aust* 15(3), Sept. 1989.
33. Ireland ML, Ott SM: Special concerns of the female athlete, *Clin Sports Med* 23(2):281-298, 2004.

34. Kenyon BJ, Loos LG: Comparing comfort and wearability between type III single-layered and double-layered EVA mouthguards, *Gen Dent* 53(4):261-264, 2005.
35. Lombardi S, Sheller B, Williams BJ: Diagnosis and treatment of dental trauma in a children's hospital, *Pediatr Dent* 20(2):112-120, 1998.
36. Newsome PR, Tran DC, Cooke MS: The role of the mouthguard in the prevention of sports-related dental injuries: a review, *Int J Paediatr Dent* 11(6):396-404, 2001.
37. Oppliger RA, Steen SA, Scott JR: Weight loss practices of college wrestlers, *Int J Sport Nutr Exerc Metab* 13(1):29-46, 2003.
38. Padilla RR: A technique for fabricating modern athletic mouthguards, *JCDA* May 33(5):399-408, 2005.
39. Padilla R, Balikov S: Sports dentistry coming of age in the 90's, *J Calif Dent Assoc* pp. 27-34, April 1993.
40. Padilla R, Dorney B, Balikov S: Mouthguards—prevention of oral injuries, *J Calif Dent Assoc* 24(3) March 1996.
41. Padilla RR, Felsenfeld AL: Treatment and prevention of alveolar fractures and related injuries, *J Craniomaxillofacial Trauma* 3(2):22-27, 1997.
42. Park: Methods of improved mouthguards, 1st International Symposium on Biomaterials, Taejon, Korea, August 1993.
43. Pinkham JR, Kohn DW: Epidemiology and prediction of sports related traumatic injuries, *Dent Clin North Am* 35(4):609-625, 1991.
44. Reed R: Origin and early history of the dental mouthpiece, *Br Dent J* 25;176(12):478-480, 1994.
45. Reports of councils and bureau's mouth protectors: 11 years on, *JADA* vol 86, June 1973.
46. Sane J: Comparison of maxillofacial and dental injuries in four contact team sports: American football, bandy, basketball, and handball, *Am J Sports Med* 16(6):647-651, 1988.
47. Stokes AN, Chapman PJ: Mouthguards, dental traumas and the 1990 all blacks, *New Zealand Journal of Sports Medicine* pp 66-67, Summer 1991.
48. Sundgot-Borgen J, Torstveit MK: Prevalence of eating disorders in elite athletes is higher than in the general population. *Clin J Sport Med* 14(1):25-32, 2004.
49. Sundgot-Borgen J, Torstveit MK, Skarderud F: Eating disorders among athletes, *Tidsskr Nor Laegeforen* 26;124(16):2126-2129, 2004.
50. Tavener M, Payne W: The effects of participation in a "health of primary schools program" on the physical fitness of children, *Aust J Sci Med Sport* 25(3):84-85, 1993.
51. Turner CH: Mouth protectors, *Br Dent J* 143(3):82-86, 1977.
52. Waked EJ, Caputo AA: Thickness and stiffness characteristics of custom-made mouthguard materials, *Quintessence Int* 36(6):462-466, 2005.
53. Walker J, Jakobsen J, Brown S: Attitudes concerning mouthguard use in 7- to 8-year-old children, *ASDC J Dent Child* 69(2):126, 207-211, 2002.
54. Upson N: Dental injuries and the attitudes of rugby players to mouthguards, *Br J Sports Med* 16(4):241-244, 1982.